
MEDICO-CHIRURGICAL SOCIETY
EDINBURGH

THE TRANSACTIONS

OF THE

MEDICO-CHIRURGICAL SOCIETY OF EDINBURGH.

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OF THE

MEDICO-CHIRURGICAL SOCIETY OF EDINBURGH.

VOL. XI.—NEW SERIES.

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1892.

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PREFACE.

The present Volume is the *Eleventh* of the *New Series*, and contains a record of the work done during the past Session.

That work, as hitherto, embraces the communication of Original Papers; the exhibition of Patients, illustrating rare and interesting forms of disease; and the exhibition of Pathological and other specimens, so essential to the proper understanding of the morbid changes which take place in the human body.

During the past Session two Extra Meetings were held. The first of these meetings was devoted to hearing a paper by Dr Harold J. Stiles on Cancer of the Breast, illustrated by Limelight and Microscopical Demonstrations. The second evening was devoted to a discussion on the Treatment of Pernicious Anæmia by the Transfusion of Human Blood, opened by Dr Brakenridge. It is hoped that such Meetings will materially increase the usefulness of the Society.

It is believed that the publication of the Transactions in this permanent form will prove a valuable contribution to medical literature, will encourage the Members to take a more active part in the work of the Society, and will tend in no small degree to increase the influence and usefulness of the Medico-Chirurgical Society of Edinburgh.

William Craig,
Editor.

October 1892.

Medico-Chirurgical Society of Edinburgh.

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135	Edwin Baily, M.B., C.M., Oban,	1883
	Alexander Black, M.B., F.R.C.P. Ed., 13 Howe Street,	1883
	Harry Melville Dunlop, M.D., F.R.C.P. Ed., 20 Abercromby Place,	1883
	* George Andreas Berry, M.B., F.R.C.S. Ed., 31 Drumsheugh Gardens,	1883
	Hamilton Wylie, M.B., C.M., 1 George Place,	1883
140	Arthur Douglas Webster, M.D., F.R.C.P. Ed., 20 Newington Road,	1883
	Robert William Philip, M.D., F.R.C.P. Ed., 4 Melville Crescent,	1883
	Joseph Carne Ross, M.D., F.R.C.P. Ed., Parsonage Nook, Withington,	1884
	William Russell, M.D., F.R.C.P. Ed., 46 Albany Street, *Secretary*,	1884
	George Dickson, M.D., F.R.C.S. Ed., 9 India Street,	1884
145	Thomas Wyld Pairman, L.R.C.P. & S. Ed., H. M. Prison, Lyttelton, New Zealand,	1884

		Date of Admission.
	Alexander Thom, M.D., C.M., Crieff,	1884
	Hugh Logan Calder, M.D., C.M., 60 Leith Walk,	1884
	James Craig Balfour, L.R.C.P. & S. Ed., West Street, Belford, Northumberland,	1884
	Frederick Anastasius Saunders, L.R.C.P. Ed., F.R.C.S. Ed., Grahamstown, South Africa,	1884
150	Wm. Richardson, M.D., F.R.C.S. Ed., Bath Lodge, Reading,	1884
	Andrew Brown, M.D., M.R.C.P. Ed., 1 Bartholomew Road, Kentish Town, London, N.W.	1884
	G. J. H. Bell, M.B., C.M., Surgeon, Bengal Army,	1884
	T. Goodall Nasmyth, M.D., D.Sc., Cupar-Fife,	1884
	Henry Hay, M.B., C.M., 7 Brandon Street,	1884
155	Thomas R. Scott, M.D., C.M., Musselburgh,	1884
	R. Milne Murray, M.B., F.R.C.P. Ed., 10 Hope Street,	1884
	A. S. Cumming, M.D., F.R.C.P. Ed., 18 Ainslie Place,	1884
	Ernest F. Neve, M.D., F.R.C.S. Ed., M.R.C.S. Eng., Hospital, Srinagar, Kashmir, N.W. India,	1884
	W. C. Greig, M.B., C.M., Tangier, Morocco,	1884
160	John Mowat, M.D., 1 Hope Park Terrace,	1885
	Skene Keith, M.B., F.R.C.S. Ed., 42 Charles Street, Berkeley Square, London, W.,	1885
	D. Noël Paton, M.D., F.R.C.P. Ed., 4 Walker Street,	1885
	George Hugh Mackay, M.B., C.M., Elgin,	1885
	Michael Dewar, M.D., C.M., 24 Lauriston Place,	1885
165	Edward M'Callum, F.R.C.S. Ed., 3 Brandon Street,	1885
	T. Edgar Underhill, M.D., F.R.C.S. Ed., Bromsgrove, Worcestershire,	1885
	John Struthers Stewart, L.R.C.P. & S. Ed., 5 Merchiston Pl.,	1885
	Allen Thomson Sloan, M.D., C.M., 22 Forth Street,	1885
	John William Ballantyne, M.D., F.R.C.P. Ed., 24 Melville St.,	1885
170	James Robertson Crease, F.R.C.S. Ed., 2 Ogle Terrace, South Shields,	1885
	George Kerr, M.B., C.M., 6 St Colme Street,	1885
	Tom Bairstow, L.R.C.P. & S. Ed., 14 Buccleuch Place	1885
	David Milligan, M.B., C.M., 11 Palmerston Place,	1885
	George Dods, M.D., L.R.C.S. Ed., 50 Great King Street,	1885
175	J. Murdoch Brown, M.B., F.R.C.P. Ed., 9 Walker Street,	1885
	Robert W. Felkin, M.D., F.R.C.S. Ed., 20 Alva Street,	1885
	S. Hale Puckle, M.B., C.M., Bishop's Castle, Shropshire,	1885
	James Haig Ferguson, M.D., F.R.C.P. Ed., M.R.C.S. Eng., 25 Rutland Street,	1885
	Charles Kennedy, M.D., C.M., 43 Minto Street,	1886
180	William Gayton, M.D., M.R.C.S. Eng., Bartram Lodge, Fleet Road, Hampstead, London, N.W.,	1886
	Reginald Ernest Horsley, M.B., F.R.C.S. Ed., 46 Heriot Row,	1886
	James Mill, M.B., M.R.C.P. Ed., 178 Ferry Road,	1886
	Robert Fraser Calder Leith, M.B., B.Sc., F.R.C.P. Ed., 129 Warrender Park Road,	1886
	Thomas M. Burn-Murdoch, M.B., C.M., 31 Morningside Road,	1886
185	Professor William Smith Greenfield, M.D., F.R.C.P. Lond. and Ed., 7 Heriot Row,	1886
	Oswald Gillespie Wood, M.D., F.R.C.S. Ed., Surgeon, Army Medical Staff, India,	1886
	James Hogarth Pringle, M.B., C.M., 256 Bath Street, Glasgow,	1886
	Nathaniel Thomas Brewis, M.B., F.R.C.P. Ed., 23 Rutland St.,	1886
	John Batty Tuke, jr., M.D., F.R.C.P. Ed., Balgreen, Murrayfield,	1886
190	David Berry Hart, M.D., F.R.C.P. Ed., 29 Charlotte Square,	1886
	Walter Scott Lang, M.D., F.R.C.S. Ed., M.R.C.S. Eng., Edinburgh,	1886
	Alfred Bell Whitton, M.B., C.M., Aberchirder,	1886
	Robert S. Aitchison, M.B., F.R.C.P. Ed., 83 Great King St.,	1887
	J. A. Armitage, M.D., C.M., 15 Waterloo Road, Wolverhampton,	1887

		Date of Admission.
195	J. Walton Hamp, L.F.P.S. Glasg., L.S.A. Lond., Wolverhampton,	1887
	William Hunter, M.D., M.R.C.S. Eng., M.R.C.P.L., 61 Wimpole Street, Cavendish Square, London, W.,	1887
	Sydney Rumboll, L.R.C.P. Ed., F.R.C.S. Ed., Hope Villa, Hillary Place, Leeds,	1887
	John Thomson, M.D., F.R.C.P. Ed., 14 Coates Crescent,	1887
	George Franklin Shiels, M.D., F.R.C.S. Ed., 229 Geary Street, San Francisco,	1887
200	T. Brown Darling, M.D., C.M., 165 Bruntsfield Place,	1887
	John Keay, M.B., M.R.C.P. Ed., Mavisbank House, Polton,	1887
	John F. Sturrock, M.B., C.M., Homewood, Broughty Ferry,	1887
	Edward Carmichael, M.D., 12 London Street,	1887
	Charles C. Teacher, M.B., C.M., 16 Newington Road,	1887
205	David W. Aitken, M.B., C.M., 17 Hatton Place,	1887
	Robert Inch, M.B., C.M., Gorebridge,	1887
	John Shaw M'Laren, M.B., F.R.C.S. Ed., 14 Walker Street,	1887
	George Mackay, M.D., F.R.C.S. Ed., M.R.C.S. Eng., 2 Randolph Place,	1887
	Henry Alexis Thomson, M.D., F.R.C.S. Ed., 2 Coates Crescent,	1887
210	David Wallace, M.B., F.R.C.S. Ed., 66 Northumberland Street,	1887
	John C. Messer, M.D., R.N., 15 Belgrave Place,	1887
	D. H. Anderson, M.B., C.M., Grange-over-Sands, Lancashire,	1887
	James Lockhart Wilson, M.B., C.M., Duns,	1888
	William Booth, F.R.C.S. Ed., 2 Minto Street,	1888
215	Professor John M'Fadyean, M.B., C.M., Royal Veterinary College, Camden Town, London,	1888
	Thomas Russell, L.F.P.S. Glasg., 27A Westmuir Street, Parkhead, Glasgow,	1888
	John Ross Home Ross, M.B., F.R.C.P. Ed., 40 York Place,	1888
	George M. Johnston, M.D., C.M., 9 Morton Street, Leith,	1888
	George Pirrie Boddie, M.B., C.M., 147 Bruntsfield Place,	1888
220	Kenneth Mackinnon Douglas, M.D., F.R.C.S. Ed., 26 Rutland Street,	1888
	George Lovell Gulland, M.D., F.R.C.P. Ed., 6 Randolph Place,	1888
	William Burns Macdonald, M.B., C.M., Port Lodge, Dunbar,	1888
	James Williamson Martin, M.D., F.R.C.P. Ed., 59 Ferry Road, Leith,	1888
	Surgeon-Captain Charles H. Bedford, M.D., D.Sc., M.R.C.S. Eng., H. M. Bengal Army, care of W. Watson & Co., 27 Leadenhall Street, London, E.C.,	1889
225	William Haldane, M.D., F.F.P.S. Glasg., Viewforth, Bridge of Allan,	1889
	John Hugh Alex. Laing, M.B., C.M., 11 Melville Street,	1889
	Harold Jalland Stiles, M.B., F.R.C.S. Ed., 5 Castle Terrace,	1889
	John Smith, M.D., M.R.C.S. Eng., Brycehall, Kirkcaldy,	1889
	Allan Cuthbertson Sym, M.D., C.M., 144 Morningside Road,	1889
230	Edmund Frederick Tanney Price, M.B., C.M., 28 Mayfield Road,	1889
	John Berry Haycraft, M.D., Sc.D., 14 Westbourne Terrace Road, London, W.,	1889
	Henry Harvey Littlejohn, M.B., F.R.C.S. Ed., 13 Victoria Road, Sheffield,	1889
	Albert Edward Morison, M.B., F.R.C.S. Ed., M.R.C.S. Eng., Brougham Terrace, Hartlepool,	1889
	William George Sym, M.D., F.R.C.S. Ed., 50 Queen Street,	1889
235	Benjamin D. C. Bell, L.R.C.P. and S. Ed., Kirkwall,	1889
	Hugh Jamieson, M.B., C.M., Hospital Inglas, Monte Video,	1889
	David Gair Braidwood, M.B., C.M., Halkirk, Caithness,	1889
	A. Home Douglas, M.B., F.R.C.P. Ed., 6 W. Maitland St.,	1889
	Alexander John Keiller, L.R.C.P. and S. Ed., 54 Northumberland Street,	1889
240	G. Keppie Paterson, M.B., M.R.C.P. Ed., 17 Forth Street,	1889
	William Stewart, M.D., F.F.P.S. Glasg., 146 Ferry Rd., Leith,	1889

		Date of Admission.
	Alfred William Hughes, M.B., F.R.C.S. Ed., M.R.C.S. Eng., Woodside, Musselburgh,	1889
	Thomas Proudfoot, M.B., M.R.C.P. Ed., 13 Lauriston Place,	1889
	Professor William H. Barrett, M.B., C.M., 21 Learmonth Ter.,	1890
245	Dawson Fyers Duckworth Turner, M.D., F.R.C.P. Ed., 7 George Square,	1890
	Edward Farr Armour, M.B., C.M., 149 Bruntsfield Place,	1890
	James Hunter, M.D., C.M., Linlithgow,	1890
	William Guy, L.R.C.P. and S. Ed., 11 Wemyss Place,	1890
	William Smith, L.R.C.P. and S. Ed., L.F.P.S. Glasg., 14 Hartington Gardens,	1890
250	Robert A. Flemming, M.B., F.R.C.P. Ed., 36 Drumsheugh Gardens,	1890
	Robert Thin, M.B., M.R.C.P. Ed., 6 Albany Street,	1890
	George M. Robertson, M.B., M.R.C.P. Ed., Morningside Royal Asylum,	1890
	John Mackintosh Balfour, M.B., M.R.C.P. Ed.,	1890
	James Hutcheson, M.D., F.R.C.S. Ed., 8 Nelson Street,	1890
255	A. Cowan Guthrie, M.B., C.M., 53 Charlotte Street, Leith,	1890
	Ralph Stockman, M.D., F.R.C.P. Ed., 12 Hope Street,	1891
	Alexander Lockhart Gillespie, M.D., F.R.C.P. Ed., 10 Walker Street,	1891
	Stewart Stirling, M.D., F.R.C.S. Ed., 6 Clifton Terrace,	1891
	Francis D. Boyd, M.B., M.R.C.P. Ed., 6 Atholl Place,	1891
260	J. J. Douglas, M.D., 22 Drummond Place,	1891
	Robert Stirling, M.B., C.M., 4 Atholl Place, Perth,	1891
	J. Y. Simpson Young, M.B., C.M., 14 Ainslie Place,	1891
	John Macpherson, M.D., F.R.C.P. Ed., Stirling District Asylum, Larbert,	1891
	John Smith, M.B., C.M., 1 Parson's Green Terrace,	1891
265	Norman Purvis Walker, M.D., F.R.C.P. Ed., 3 Torphichen St.,	1891
	Charles Templeman, M.D., C.M., 8 Airlie Place, Dundee,	1891
	Simson C. Fowler, M.B., C.M., Juniper Green,	1892
	William Towers-Smith, M.R.C.S. Eng., 24 Devonshire Street, Portland Place, London, W.,	1892
	Alexander Miles, M.D., F.R.C.S. Ed., 27 St Bernard's Crescent,	1892
270	Robert Abernethy, M.B., M.R.C.P. Ed., 18 Alva Street,	1892
	Robert Dundas Helm, M.D., C.M., 8 Brunswick St., Carlisle,	1892
	Frederick Thomas Anderson, L.R.C.P. & S. Ed., L.F.P.S. Glasg., Standford, Liphook, Hampshire,	1892
	Arthur Logan Turner, M.B., F.R.C.S. Ed., 19 Rutland Street,	1892
	T. Herbert Littlejohn, M.B., C.M., 24 Royal Circus,	1892
275	G. Matheson Cullen, M.D., C.M., 48 Lauriston Place,	1892

NON-RESIDENT.

	Arthur Edward Turnour, M.D., M.R.C.S. Eng., *Denbigh*,	1843
	W. Ord M'Kenzie, M.D., L.R.C.S. Ed., *London*,	1845
	W. Judson Van Someren, M.D., L.R.C.S. Ed., *Redhill, Surrey*,	1845
	William H. Lowe, M.D., F.R.C.P. Ed., *Wimbledon*,	1845
280	George Skene Keith, M.D., F.R.C.P. Ed., *Currie*,	1845
	Veitch Sinclair, L.R.C.P. and S. Ed., *London*,	1850
	Archibald Hall, M.D., *Montreal*,	1853
	W. Overend Priestley, M.D., LL.D., F.R.C.P. Ed., *London*,	1854
	Horatio Robinson Storer, M.D., *Newport, Rhode Island, U.S.*,	1855
285	James C. Howden, M.D., *Montrose*,	1856
	Thomas Skinner, M.D., L.R.C.S. Ed., *London*,	1856
	Professor William Smoult Playfair, M.D., LL.D., F.R.C.P.L., *London*,	1857
	J. Ivor Murray, M.D., F.R.C.S. Ed., *Scarboro'*,	1857
	Andrew Scott Myrtle, M.D., L.R.C.S. Ed., *Harrogate*,	1859
290	Robert Foulis, M.D., F.R.C.S. Ed., *Cupar-Fife*,	1859
	Francis Robertson Macdonald, M.D., *Inveraray*,	1860

		Date of Admission.
	Professor John Young, M.D., *University of Glasgow*, . .	1860
	George Thin, M.D., L.R.C.S. Ed., *London*, . . .	1861
	J. Cecil Phillippo, M.D., *Kingston, Jamaica*, . . .	1861
295	Professor William Stephenson, M.D., F.R.C.S. Ed., *Aberdeen*,	1861
	J. S. Beveridge, M.R.C.P. Lond., F.R.C.S. Ed., *Edenbridge*, .	1861
	David Yellowlees, M.D., LL.D., F.F.P.S. Glasg., *Glasgow*, .	1862
	Prof. Arthur Gamgee, M.D., F.R.C.P. Ed., F.R.S., *London*, .	1864
	Professor John Cleland, M.D., LL.D., *The University, Glasgow*,	1864
300	R. B. Finlay, M.D., Q.C., *Middle Temple, London*, . .	1864
	Stanley Lewis Haynes, M.D., M.R.C.S. Eng., *Malvern*, .	1864
	James Watt Black, M.D., F.R.C.P.L., *London*, . .	1865
	David Brodie, M.D., *Canterbury*, ...	1865
	Peter Maury Deas, M.B., L.R.C.S. Ed., *Exeter*, . .	1868
305	Professor J. G. M'Kendrick, M.D., LL.D., F.R.C.P. Ed., *University, Glasgow*, ...	1870
	Professor Lawson Tait, M.D., F.R.C.S. Ed. and Eng., LL.D., *Birmingham*, ...	1870
	J. G. Sinclair Coghill, M.D., F.R.C.P. Ed., *Ventnor*, . .	1870
	James Johnston, M.D., F.R.C.S. Ed., *London*, . .	1871
	J. William Eastwood, M.D., M.R.C.P.L., *Darlington*, .	1871
310	Professor J. Bell Pettigrew, M.D., LL.D., F.R.C.P. Ed., *University of St Andrews*, ...	1873
	J. Johnson Bailey, M.D., F.R.C.S. Ed., *Marple*, . .	1874
	John Aymers Macdougall, M.D., F.R.C.S. Ed., *France*, .	1875
	Thomas John Maclagan, M.D., M.R.C.P.L., *London*, . .	1875
	Dr Groesbeck, *Cincinnati*, ...	1875
315	Professor David James Hamilton, M.B., F.R.C.S. Ed., *Aberdeen University*, ...	1876
	J. Moolman, M.B., C.M., *Cape of Good Hope*, . . .	1877
	Robert Somerville, M.D., F.R.C.S. Ed., *Galashiels*, . .	1877
	Graham Steell, M.D., F.R.C.P.L., *Manchester*, . .	1877
	Frederick William Barry, M.D., D.Sc., *London*, . .	1878
320	John Brown, M.D., F.R.C.S. Eng., *Burnley*, . . .	1878
	Keith Norman Macdonald, M.D., F.R.C.P. Ed., *Skye*, .	1880
	John Home Hay, M.D., M.R.C.S. Eng., *Alloa*, . .	1880
323	John Mackay, M.D., L.R.C.S. Ed., *Aberfeldy*, . . .	1881

ORDINARY MEMBERS

ARRANGED ALPHABETICALLY.

RESIDENT.

	Dr R. Abernethy, 18 Alva Street, ...	1892
	Dr J. O. Affleck, 38 Heriot Row, ...	1871
	Dr R. S. Aitchison, 83 Great King Street, . . .	1887
	Dr D. Aitken, 17 Hatton Place, ...	1887
5	Dr D. H. Anderson, Grange-over-Sands, Lancashire, . .	1887
	Dr Fred. T. Anderson, Standford, Liphook, Hampshire, .	1892
	Dr James Andrew, 2 Atholl Crescent, ...	1869
	Professor Annandale, 34 Charlotte Square, . . .	1863
	Dr Archibald, Woodhouse-Eaves, Loughborough, . .	1882
10	Dr J. A. Armitage, 15 Waterloo Road, Wolverhampton, .	1887
	Dr E. F. Armour, 149 Bruntsfield Place, . . .	1890
	Dr W. Badger, Penicuik, ...	1882
	Dr Edwin Baily, Oban, ...	1883
	Tom Bairstow, Esq., 14 Buccleuch Place, . . .	1885
15	Dr Andrew Balfour, Portobello, ...	1874
	Dr J. H. Balfour, Portobello, ...	1881
	Dr G. W. Balfour, 17 Walker Street, ...	1874
	Dr James Craig Balfour, West Street, Belford, Northumberland,	1884
	Dr J. M. Balfour, ...	1890

		Date of Admission.
20	Dr Thomas Balfour, 51 George Square,	1856
	Dr Alexander Ballantyne, Dalkeith,	1872
	Dr J. W. Ballantyne, 24 Melville Street,	1885
	Dr A. H. Freeland Barbour, 8 Melville Crescent,	1881
	Professor W. H. Barrett, 21 Learmonth Terrace,	1890
25	Surgeon-Captain C. H. Bedford, M.D., H. M. Bengal Army, Indian Medical Service, care of W. Watson & Co., 27 Leadenhall Street, London, E C.,	1889
	Dr Benjamin D. C. Bell, Kirkwall,	1889
	Dr G. J. H. Bell, Surgeon, Bengal Army,	1884
	Joseph Bell, Esq., 2 Melville Crescent, *President*,	1862
	G. H. Bentley, Esq., Kirkliston,	1877
30	Dr G. A. Berry, 31 Drumsheugh Gardens,	1883
	Dr Alexander Black, 13 Howe Street,	1883
	Dr W. T. Black, 2 George Square,	1877
	Dr Robert H. Blaikie, 42 Minto Street,	1883
	Dr Bleloch, 2 Lonsdale Terrace,	1871
35	Dr G. P. Boddie, 147 Bruntsfield Place,	1888
	William Booth, Esq., 2 Minto Street,	1888
	Dr F. D. Boyd, 6 Atholl Place.	1891
	Dr D. G. Braidwood, Halkirk, Caithness,	1889
	Dr Brakenridge, 10 St Colme Street, *Vice-President*,	1865
40	Dr Byrom Bramwell, 23 Drumsheugh Gardens,	1876
	Dr N. T. Brewis, 23 Rutland Street,	1886
	Dr Brown, 1 Bartholomew Road, Kentish Town, London, N.W.,	1884
	Dr J. Graham Brown, 16 Ainslie Place,	1878
	Dr J. Macdonald Brown, 12 Cumin Place,	1883
45	Dr J. Murdoch Brown, 9 Walker Street,	1885
	Dr Alexander Bruce, 13 Alva Street,	1883
	Dr Buist, 1 Clifton Terrace,	1877
	Dr T. M. Burn-Murdoch, 31 Morningside Road,	1886
	Dr Cadell, 22 Ainslie Place,	1870
50	Dr Francis M. Caird, 21 Rutland Street,	1883
	Dr H. L. Calder, 60 Leith Walk,	1884
	Dr W. Watson Campbell, Duns,	1877
	Dr Cappie, 37 Lauriston Place,	1855
	Dr Edward Carmichael, 12 London Street,	1887
55	Dr J. Carmichael, 22 Northumberland Street,	1870
	Dr C. W. Cathcart, 8 Randolph Crescent,	1883
	Dr T. F. S. Caverhill, 8A Abercromby Place,	1884
	Professor John Chiene, 26 Charlotte Square,	1867
	Dr Church, 36 George Square,	1876
60	Dr Clouston, Tipperlinn House, Morningside Place,	1861
	Dr A. R. Coldstream, Florence, Italy,	1878
	Dr John Connel, Peebles,	1876
	Dr Cotterill, 24 Manor Place,	1878
	Dr William Craig, 71 Bruntsfield Place,	1869
65	Dr J. R. Crease, 2 Ogle Terrace, South Shields,	1885
	Dr Halliday Croom, 25 Charlotte Square,	1870
	Dr G. Matheson Cullen, 48 Lauriston Place,	1892
	Dr A. S. Cumming, 18 Ainslie Place,	1884
	Dr R. J. B. Cunynghame, 18 Rothesay Place	1868
70	Dr T. B. Darling, 165 Bruntsfield Place,	1887
	Dr M. Dewar, 24 Lauriston Place,	1885
	Dr Archibald Dickson, Hartree House, Biggar,	1871
	Dr George Dickson, 9 India Street,	1884
	Dr George Dods, 50 Great King Street,	1885
75	Dr Halliday Douglas, 30 Melville Street,	1842
	Dr A. Home Douglas, 6 West Maitland Street,	1889
	Dr J J. Douglas, 22 Drummond Place,	1891
	Dr Kenneth M. Douglas, 26 Rutland Street,	1888
	Dr William B. Dow, Dunfermline,	1879
80	Dr John Duncan, 8 Ainslie Place,	1868
	Dr Kirk Duncanson, 22 Drumsheugh Gardens,	1871

		Date of Admission
	Dr H. M. Dunlop, 20 Abercromby Place,	1883
	Dr J. Dunsmure, 53 Queen Street,	1872
	Dr R. W. Felkin, 20 Alva Street,	1885
85	Dr J. Haig Ferguson, 25 Rutland Street,	1885
	Dr W. A. Finlay, St Helen's, Russell Place, Trinity,	1875
	Dr Andrew Fleming, 8 Napier Road,	1880
	Dr R. A. Fleming, 36 Drumsheugh Gardens,	1890
	Dr Foulis, 34 Heriot Row,	1875
90	Dr Simson C. Fowler, Juniper Green,	1892
	Dr F. W. Dyce Fraser, Gorton House, Lasswade,	1883
	Dr John Fraser, 19 Strathearn Road,	1878
	Professor Thomas R. Fraser, 13 Drumsheugh Gardens,	1865
	Dr R. Freeland, Broxburn,	1879
95	Dr Garland, 35 Charlotte Street, Leith,	1873
	Dr W. Gayton, Bartram Lodge, Fleet Road, Hampstead, London, N.W.,	1886
	Dr G. A. Gibson, 17 Alva Street,	1880
	Dr A. Lockhart Gillespie, 10 Walker Street,	1891
	G. R. Gilruth, Esq., 48 Northumberland Street,	1869
100	Dr J. Allan Gray, 107 Ferry Road,	1879
	Professor Greenfield, 7 Heriot Row,	1886
	Dr David Greig, 38 Coates Gardens,	1854
	Dr W. C. Greig, Tangier, Morocco,	1884
	Dr G. L. Gulland, 6 Randolph Place,	1888
105	His Excelleney Dr R. H. Gunning, 12 Addison Crescent, West Kensington, London, W.,	1846
	Dr A. C. Guthrie, 53 Charlotte Street, Leith,	1890
	Dr William Guy, 11 Wemyss Place,	1890
	Dr John Haddon, Marsh House, Canonbie,	1883
	Dr William Haldane, Viewforth, Bridge of Allan,	1889
110	Dr J. W. Hamp, Wolverhampton,	1887
	Professor A. W. Hare, 258 Oxford Road, Manchester,	1883
	Dr D: Berry Hart, 29 Charlotte Square,	1886
	Dr Henry Hay, 7 Brandon Street,	1884
	Dr J. Berry Haycraft, 14 Westbourne Terrace Road, London, W.,	1889
115	Dr R. Dundas Helm, 8 Brunswick Street, Carlisle,	1892
	Dr John Henderson, 7 John's Place, Leith,	1848
	Dr J. W. B. Hodsdon, 52 Melville Street,	1883
	Dr R. E. Horsley, 46 Heriot Row,	1886
	Dr A. W. Hughes, Woodside, Musselburgh,	1889
120	Dr George Hunter, 33 Palmerston Place,	1876
	Dr James Hunter, St Catherine's, Linlithgow,	1890
	Dr W. Hunter, 61 Wimpole Street, Cavendish Square, London, W.,	1887
	Dr Husband, 4 Royal Circus,	1849
	Dr J. Hutcheson, 8 Nelson Street,	1890
125	Dr Robert Inch, Gorebridge,	1887
	Dr W. Wotherspoon Ireland, Prestonpans,	1883
	Dr James, 44 Melville Street,	1877
	Dr Allan Jamieson, 35 Charlotte Square,	1876
	Dr Hugh Jamieson, Hospital Inglas, Monte Video,	1889
130	Dr James Jamieson, 43 George Square,	1877
	Dr G. M. Johnston, 9 Morton Street, Leith,	1888
	Dr R. M'Kenzie Johnston, 44 Charlotte Square,	1883
	Dr J. Carlyle Johnstone, Melrose Asylum, Melrose,	1882
	Dr J. Keay, Mavisbank House, Polton,	1887
135	Dr A. J. Keiller, 54 Northumberland Street,	1889
	Dr Skene Keith, 42 Charles St., Berkeley Square, London, W.,	1885
	Dr C. Kennedy, 43 Minto Street,	1886
	Dr George Kerr, 6 St Colme Street,	1885
	Dr J. H. A. Laing, 11 Melville Street,	1889
140	Dr W. Scott Lang, Edinburgh,	1886
	Dr Robert Lawson, 24 Mayfield Terrace,	1881
	Dr R. F. C. Leith, 129 Warrender Park Road,	1886

		Date of Admission.
	Dr George Leslie, Falkirk,	1881
	Dr Linton, 60 George Square,	1863
145	Dr Littlejohn, 24 Royal Circus,	1853
	Dr Harvey Littlejohn, 13 Victoria Road, Sheffield,	1889
	Dr Herbert Littlejohn, 24 Royal Circus,	1892
	Dr Lucas, Dalkeith,	1875
	Dr R. A. Lundie, 55A Grange Road,	1883
150	Dr P. M'Bride, 16 Chester Street,	1879
	Dr E. M'Callum, 3 Brandon Street,	1885
	Dr W. Burn Macdonald, Port Lodge, Dunbar,	1888
	Professor J. M'Fadyean, Royal Veterinary College, Camden Town, London,	1888
	John M'Gibbon, Esq., 55 Queen Street,	1868
155	Dr MacGillivray, 11 Rutland Street,	1877
	Dr G. Mackay, 2A Gilmore Place,	1878
	Dr George Mackay, 2 Randolph Place,	1887
	Dr G. H. Mackay, Elgin,	1885
	Professor Sir Douglas Maclagan, 28 Heriot Row,	1834
160	Dr J. S. M'Laren, 14 Walker Street,	1887
	Dr P. H. Maclaren, 1 Drumsheugh Gardens, *Vice-President*,	1868
	Dr Roderick M'Laren, 23 Portland Square, Carlisle,	1882
	Dr John Macpherson, Stirling District Asylum, Larbert,	1891
	Dr J. W. Martin, 59 Ferry Road, Leith,	1888
165	Dr A. Matthew, Corstorphine,	1882
	Dr D. Menzies, 20 Rutland Square,	1878
	Dr J. C. Messer, 15 Belgrave Place,	1887
	Dr A. Miles, 27 St Bernard's Crescent,	1892
	Dr J. Mill, 178 Ferry Road,	1886
170	A. G. Miller, Esq., 7 Coates Crescent,	1867
	Dr D. Milligan, 11 Palmerston Place,	1885
	Sir Arthur Mitchell, 34 Drummond Place,	1859
	Dr Moir, 52 Castle Street,	1836
	Dr Alexander Moir, 30 Buccleuch Place,	1876
175	Dr Albert Edward Morison, Hartlepool,	1889
	Dr J. Rutherford Morison, 14 Saville Row, Newcastle-on-Tyne,	1882
	Dr John Mowat, 1 Hope Park Terrace,	1885
	Dr Claud Muirhead, 30 Charlotte Square, *Vice-President*,	1866
	Dr R. Milne Murray, 10 Hope Street,	1884
180	Dr A. D. Leith Napier, 67 Grosvenor Street, Grosvenor Square, London, W.,	1879
	Dr T. Goodall Nasmyth, Cupar-Fife,	1884
	Dr E. F. Neve, Dispensary, Srinagar, Kashmir, N.W. India,	1884
	Dr H. Newcombe, 5 Dalrymple Crescent,	1883
	Dr P. Orphoot, 113 George Street,	1865
185	Dr T. W. Pairman, H. M. Prison, Lyttelton, New Zealand,	1884
	Dr Paterson, 4 Coates Crescent,	1847
	Dr G. Keppie Paterson, 17 Forth Street,	1889
	Dr D. Noël Paton, 4 Walker Street,	1885
	Dr Peddie, 15 Rutland Street,	1842
190	Dr J. A. Philip, Rue Victor Hugo, Boulogne-Sur-Mer,	1878
	Dr R. W. Philip, 4 Melville Crescent,	1883
	Dr Playfair, 5 Melville Crescent,	1874
	Dr Edmund Price, 28 Mayfield Road,	1889
	Dr J. H. Pringle, 256 Bath Street, Glasgow,	1886
195	Dr T. Proudfoot, 13 Lauriston Place,	1889
	Dr S. Hale Puckle, Bishop Castle, Shropshire,	1885
	Dr Rattray, Portobello,	1874
	Dr William Richardson, Bath Lodge, Reading,	1884
	Dr James Ritchie, 14 Charlotte Square,	1873
200	Dr R. Peel Ritchie, 1 Melville Crescent,	1862
	Dr Argyll Robertson, 18 Charlotte Square,	1861
	Dr G. M. Robertson, Morningside Royal Asylum,	1890
	Dr Ronaldson, 3 Bruntsfield Terrace,	1877

		Date of Admission.
	Dr J. Maxwell Ross, Calderbank, Maxwelltown, Dumfries, .	1882
205	Dr Joseph C. Ross, Parsonage Nook, Withington, . .	1884
	Dr Home Ross, 40 York Place, ...	1888
	Dr S. Rumboll, Hope Villa, Hillary Place, Leeds, . .	1887
	Thomas Russell, Esq., 27A Westmuir St., Parkhead, Glasgow,	1888
	Dr William Russell, 46 Albany Street, *Secretary*, . .	1884
210	Professor Rutherford, 14 Douglas Crescent, . . .	1866
	Dr F. A. Saunders, Grahamstown, South Africa, . .	1884
	Dr Thomas R. Scott, Musselburgh, ...	1884
	Dr Andrew Semple, 10 Forres Street, ...	1883
	Dr John Shand, 34 Albany Street, ...	1878
215	C. H. E. Sheaf, Esq., Toowoomba, Queensland, Australia, .	1871
	Dr G. F. Shiels, 229 Geary Street, San Francisco, . .	1887
	Dr W. H. Shirreff, Melbourne, Australia, . . .	1883
	Dr J. Sibbald, 3 St Margaret's Road, ...	1859
	Professor Simpson, 52 Queen Street, ...	1859
220	Dr A. T. Sloan, 22 Forth Street, ...	1885
	Dr Andrew Smart, 20 Charlotte Square, . . .	1865
	Dr G. D. Smith, 148 Ferry Road, . . .	1877
	Dr John Smith, 11 Wemyss Place, ...	1856
	Dr John Smith, Brycehall, Kirkcaldy, . . .	1889
225	Dr John Smith, 1 Parson's Green Terrace, . . .	1891
	Dr Towers-Smith, 24 Devonshire St., Portland Pl., London, W.,	1892
	Dr William Smith, 14 Hartington Gardens, . . .	1890
	Professor Grainger Stewart, 19 Charlotte Square, . .	1861
	Dr J. S. Stewart, 5 Merchiston Place, ...	1885
230	Dr W. Stewart, Kirkwall, ...	1879
	Dr William Stewart, 146 Ferry Road, Leith, . . .	1889
	Dr H. J. Stiles, 5 Castle Terrace, ...	1889
	Dr R. Stirling, 4 Athole Place, Perth, . . .	1891
	Dr S. Stirling, 6 Clifton Terrace, ...	1891
235	Dr R. Stockman, 12 Hope Street, ...	1891
	Dr John Strachan, Dollar, ...	1867
	Dr J. F. Sturrock, Homewood, Broughty Ferry, . .	1887
	Dr Allan C. Sym, 144 Morningside Road, . . .	1889
	Dr William G. Sym, 50 Queen Street, ...	1889
240	Dr Johnson Symington, 2 Greenhill Park, . . .	1878
	Dr W. Taylor, 12 Melville Street, ...	1871
	Dr C. C. Teacher, 16 Newington Road, . . .	1887
	Dr C. Templeman, 8 Airlie Place, Dundee, . . .	1891
	Dr C. H. Thatcher, 13 Albany Street, ...	1876
245	Dr R. Thin, 6 Albany Street, ...	1890
	Dr Alexander Thom, Crieff, ...	1884
	Dr Alexis Thomson, 2 Coates Crescent, . . .	1887
	Dr John Thomson, 14 Coates Crescent, . . .	1887
	Dr J. Stitt Thomson, Dalkeith, ...	1877
250	Dr Francis Troup, 1 Minto Street, *Treasurer*, . . .	1883
	Dr Batty Tuke, 20 Charlotte Square, ...	1864
	Dr J. Batty Tuke, jr., Balgreen, Murrayfield, . . .	1886
	Dr Dawson F. D. Turner, 7 George Square, . . .	1890
	Dr Logan Turner, 19 Rutland Street, ...	1892
255	Dr R. S. Turner, Keith, ...	1867
	Dr Underhill, 8 Coates Crescent, ...	1872
	Dr T. Edgar Underhill, Broomsgrove, Worcestershire, .	1885
	Dr Norman Walker, 3 Torphichen Street, . . .	1891
	Dr D. Wallace, 66 Northumberland Street, . . .	1887
260	Dr P. H. Watson, 16 Charlotte Square, . . .	1856
	Dr W. Watson, 34 Fountainhall Road, . . .	1862
	Dr A. D. Webster, 20 Newington Road, . . .	1883
	Dr A. B. Whitton, Aberchirder, ...	1886
	Dr J. Lockhart Wilson, Duns, ...	1888
265	J. L. Wilson, Esq., 4 Buccleuch Place, . . .	1883
	Dr T. D. Wilson, 10 Newington Road, ...	1880
	Dr Oswald G. Wood, India, ...	1886

		Date of Admission.
	Dr Russell E. Wood, 9 Darnaway Street,	1883
	Dr G. Sims Woodhead, Beverley, Nightingale Lane, Balham, London, S.W.,	1883
270	Dr Strethill Wright, 107 Chatham Street, Liverpool,	1871
	Dr Hamilton Wylie, 1 George Place,	1883
	Dr John Wyllie, 1 Melville Street,	1868
	Dr James Young, 14 Ainslie Place,	1859
	Dr J. Y. Simpson Young, 14 Ainslie Place,	1891
275	Dr P. A. Young, 25 Manor Place,	1870

NON-RESIDENT.

	Dr J. J. Bailey, *Marple, Cheshire*,	1874
	Dr F. W. Barry, *London*,	1878
	Dr J. S. Beveridge, *Edenbridge*,	1861
	Dr J. W. Black, *London*,	1865
280	Dr Brodie, *Canterbury*,	1865
	Dr John Brown, *Burnley*,	1878
	Professor Cleland, *Glasgow*,	1864
	Dr Coghill, *Ventnor*,	1870
	Dr P. M. Deas, *Exeter*,	1868
285	Dr J. W. Eastwood, *Darlington*,	1871
	Dr R. B. Finlay, Q.C., *Middle Temple, London*,	1864
	Dr Foulis, *Cupar-Fife*,	1859
	Professor Gamgee, *St-Leonards-on-Sea*,	1863
	Dr Groesbeck, *Cincinnati*,	1875
290	Dr Archibald Hall, *Montreal*,	1853
	Professor D. J. Hamilton, *Aberdeen University*,	1876
	Dr J. H. Hay, *Alloa*,	1880
	Dr Stanley Haynes, *Malvern*,	1864
	Dr J. S. Howden, *Montrose*,	1856
295	Dr James Johnston, *London*,	1871
	Dr George Keith, *Currie*,	1845
	Dr Lowe, *Wimbledon*,	1845
	Dr F. R. Macdonald, *Inveraray*,	1860
	Dr K. N. Macdonald, *Skye*,	1880
300	Dr John A. Macdougall, *France*,	1875
	Dr John Mackay, *Aberfeldy*,	1881
	Professor M'Kendrick, *Glasgow*,	1870
	Dr W. O. M'Kenzie, D.I.G.H., *London*,	1845
	Dr T. J. Maclagan, *London*,	1875
305	Dr J. Moolman, *Cape of Good Hope*,	1877
	Dr J. Ivor Murray, *Scarboro'*,	1857
	Dr Andrew Myrtle, *Harrogate*,	1859
	Professor Bell Pettigrew, *St Andrews*,	1873
	Dr Phillippo, *Kingston, Jamaica*,	1860
310	Professor W. S. Playfair, *London*,	1857
	Dr Priestley, *London*,	1854
	Dr Sinclair, *London*,	1850
	Dr T. Skinner, *London*,	1856
	Dr Van Someren, *Redhill, Surrey*,	1845
315	Dr Somerville, *Galashiels*,	1877
	Dr Graham Steell, *Manchester*,	1877
	Professor Stephenson, *Aberdeen*,	1861
	Dr H. R. Storer, *Newport, Rhode Island, U.S.*,	1855
	Professor Lawson Tait, LL.D., *Birmingham*,	1870
320	Dr Thin, *London*,	1861
	Dr Turnour, *Denbigh*,	1843
	Dr Yellowlees, *Gartnavel Asylum, Glasgow*,	1862
323	Professor John Young, *Glasgow*,	1860

N.B.—*Members are requested to communicate with the Secretaries if they discover any errors or omissions in the List, and also to intimate all changes in their addresses.*

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ERRATUM.

Page 130, line 31, *for* "Cyclopædia" *read* "Cyropædia."

TRANSACTIONS

OF

THE MEDICO-CHIRURGICAL SOCIETY OF EDINBURGH,

FOR SESSION LXXI., 1891-92.

Meeting I.—November 4, 1891.

Professor A. R. Simpson, *President, in the Chair.*

I. VALEDICTORY ADDRESS.

By Professor A. R. Simpson, M.D., President.

Gentlemen,—On rising for the last time to address you from the Chair, it becomes me to offer you my hearty thanks for the kindness that placed me in this honourable position. Many men whose names Edinburgh will always cherish as the ornaments of our profession have filled the office, and its most ambitious member might well be proud to become President of the Medico-Chirurgical Society of Edinburgh. But I wish to thank you the more because I feel that in choosing me to be your President you not only conferred on me an honour, you at the same time bestowed on me a boon; for in laying on me the obligation to take the Chair at your meetings, you gave me the opportunity of hearing many important papers and discussions, and of seeing a variety of interesting patients and preparations illustrative of conditions with which I had little likelihood of becoming otherwise acquainted.

The Society an Universitas of Medicine.

In these days of rapidly-increasing specialism, when with the advance of every ancillary science the progress of our Art in its various departments makes it impossible for one to be profi-

cient in each, there is a growing tendency for us to gather in our special groups, and so to concentrate attention on our individual subjects as to fall out of familiarity with all the rest. It is one of the happy and fruitful offices of this Society to call us from our divergent spheres, and gather us round themes of common interest to all. It is in its meetings that we get the clearest impression of the advances that are being made in the several directions along which Medicine progresses, and those who are working in one direction often get not only stimulus but guidance in listening to the reports of those who are working in another.

As an illustration I may refer to the practice of flap-splitting in the repair of injuries of the perineum. As is well known, the wide acceptance of this procedure is largely due to the genius and energy of a gynecologist, of whose great achievements Edinburgh must be ever proud—I mean Professor Lawson Tait of Birmingham. But some of our co-workers, especially in Germany, were led to adopt it from the description given of the operation in Hart and Barbour's widely-circulated *Manual of Gynecology;* and what I wish to indicate just now is, that it was from hearing in this Society a paper by Dr Joseph Bell in 1876, in which he described the successful closure of the pudenda in a distressing case of vesico-vaginal fistula, that it occurred to me that perineal damages might all be repaired by simply dissecting up flaps of mucous membrane to secure sufficient raw surface for re-union, instead of paring away the superficial tissues, as had been the practice previously. Mr Bell told us, in the subsequent discussion, that he had probably got the idea from a statement by Dr John Duncan, at some previous meeting, that he had adopted the principle in operating for the cure of artificial anus. It is thus that one mind stimulates another, and the success that has been achieved in one direction leads to good results in others also.

I.—Comprehensiveness shown in the Presidential Succession.

From three different points of view the comprehensive character of the Society impresses itself on one. *First*, that in succession to a Dentist, who had been preceded by a pure Physician, you chose an Obstetrician for your President, and that he in turn should be succeeded by a member who has won his laurels in the domain of Surgery—that, surely, is a very striking evidence of the wide variety of interests that are represented here.

II.—The Death-Roll.

Again, when we scan the death-roll of the last two years, we are struck by the same note of comprehensiveness. From the list of our Foreign Corresponding Members there have fallen the

names of Jacob Bigelow, the illustrious Professor of Surgery in Harvard University, Boston, and of Fordyce Barker, of New York, who, if he had not been the foremost Obstetrician in America, would have been placed in the very front rank of her Physicians, and on whom not only the colleges of his own country but the two leading universities of Scotland delighted to confer their honorary degree of LL.D. Of Corresponding Members in the United Kingdom, we have lost Sir William Gull, foremost among the London Physicians of his day; Professor John Marshall, the Surgeon who presided with so much dignity and distinction over the affairs of the General Medical Council; and a great Obstetrician in the person of Dr James Matthews Duncan. This last name comes closest to us here, and the mention of it now touches in many hearts tender chords of admiration and affection. My first memory of Dr Duncan goes back to a school vacation when he gave my brother and me lessons in rowing on the Holy Loch; and my next to a visit he paid to Bathgate, along with Dr Thomas Keith, to report the results of operations which my uncle had been out the week before performing on some pigs in my father's premises. What made that visit specially memorable was that, on the way out to see our famous Academy, a game of leap frog was started on the road-side, and for once in my life I had the delight of clearing a back high enough, and shoulders broad enough, to satisfy the vaulting ambition of any school-boy. That must have been in 1849. When I came to Edinburgh as a student in 1850, Duncan still kept coming to 52 Queen Street to consult books in the library; but already a coolness had set in between him and his "chief" that settled into an estrangement, which was, I fancy, a life-long sorrow for them both. They were men of very diverse temperament—differing from each other as does the eager, heaven-searching Greek from the determined, earth-subduing Roman. But they were both of them splendid workers; and, happily for us, the work of each abides. It was in this Society that Matthews Duncan communicated to the profession many of his most valuable researches. For years he took a prominent part in its debates; as a Member of Council and a Vice-President he took his share in the management of its affairs; and on his removal to London, the Society expressed its high appreciation of his worth by enrolling him in the honoured list of its Corresponding Members. His memory will long be cherished among us, and for many generations the treatises on Midwifery will make mention of his name.

The losses from among our Ordinary Members still illustrate the variety of minds that are wont to assemble here. Dr William Menzies for more than half a century conducted one of the most extensive practices that has almost ever fallen to the lot of any Edinburgh doctor. "I've been three times out of my bed this week at confinement cases, and I have sixty visits to pay to-day," he would

say to one who wished to detain him of a morning; and off he would go from street to street and stair to stair, never hasting, never resting, carrying confidence and comfort into many a home. That he was able to a green old age to go through such unremitting toil he attributed to the practice—which he had adopted in early life—of total abstinence from all intoxicants; and I do not doubt that he was right. Dr James Struthers was endowed with gifts that would have ensured him a high place among the specialists if he had not devoted himself to the work of a general practitioner. As it was, he rose to a position of eminence, and those who had occasion to meet him were not surprised at the confidence with which he inspired his patients when they saw the zest with which he continued to study diseases not only in their practical but their scientific aspects. It was with a generous and characteristic self-effacement that in his will he bequeathed to the Royal College of Physicians £1000, the interest of which should go, not to perpetuate his own memory, but to increase the value of a prize that bears another Fellow's name. Dr Robert Bruce was also a practitioner of wide experience, who sometimes gave the benefit of his observations to our Societies, especially when the discussions turned on the diseases of infants. Dr William Ziegler, who was for many years in succession to his father one of the Physicians to the Maternity Hospital, did good work in connexion with that Institution. Though of delicate frame, he lived an active life and added honour to a name already honoured in Obstetrics. Dr William Wilson had not long graduated, and had just begun a career of high promise in assisting his father as a dentist, when he was taken away. Besides these five Ordinary Members, four have died who were not resident in Edinburgh, viz., Dr Andrew Graham, Fleet Surgeon, R.N.; Dr Peter Gordon, of Juniper Green; Dr Thomas Sheriff; and Dr Walter Weir, of London.

III.—Diversity of Contributors to Transactions.

The diversity of pursuit of your successive Presidents, and the varied associations that gather round the names of our departed brethren, alike declare our Society to be a kind of Universitas of Medicine. But, *thirdly*, the wide range of its interests comes still more clearly into view when we glance at the names of those who have contributed papers to our Transactions, or exhibited patients, preparations and specimens, or taken part in our debates. Surgery is represented by Professor Annandale, Messrs Caird and Cathcart, Professor Chiene, Messrs Kenneth Douglas, Duncan, Scott Lang, Shaw Maclaren, Miles, Miller, Alexis Thomson, and Wallace; Medicine, by Drs Affleck, Brakenridge, Bramwell, Felkin, James, Philip, A. Smart, and Professor Grainger Stewart; Pathology, by Drs Barrett and Bruce, Professor Greenfield and Dr Russell; Psychiatrics, by Dr Clouston; Gynecology, by Drs Ballantyne, Brewis,

Halliday Croom, and Berry Hart; Pædiatrics, by Drs Carmichael, Playfair, and John Thomson; Dermatology, by Dr Allan Jamieson; Ophthalmology, by Messrs Berry and W. G. Sym; Otology, by Drs Horsley and M'Bride; Dentistry, by Dr John Smith; Public Health, by Dr Littlejohn and Dr Harvey Littlejohn; Anatomy and Physiology, by Dr Haycraft and Mr Hughes: whilst of our Resident Members engaged in general practice, Drs J. M. Balfour, Boddie, Edie, Gulland, Kennedy, Lundie, Burn Murdoch, James Ritchie, Ronaldson, Shand, Alan Sym, Taylor, and Troup, and of our Non-resident Members, Dr Andrew Balfour of Portobello, Dr Bedford of the Bengal Medical Service, Dr Begg of Hankow, Dr Leslie of Falkirk, and, last in this alphabetical list though not least in the importance of his communication, Dr Strachan of Dollar have all contributed in various ways to increase the value of our two volumes of Transactions.

Three Outstanding Meetings.

It were too long to attempt even to enumerate the subjects that have been brought before us by these gentlemen during the last two years. But three of our meetings were specially well attended because of the wide interest attaching to the topics that were brought forward for discussion, and deserve to be recalled on this occasion.

I. The Physiology of Education.

The relation of school work to the health of children was brought before us in a very thoughtful and suggestive address by Dr Strachan of Dollar, who was well able to deal with the subject from his long observation of the health conditions of young people in that seat of education. Besides the observations that were made by our own members, the subject was further discussed by several of the leading educationalists of Edinburgh, with whose presence we were favoured on that occasion. The interest that the matter evoked was shown not only by the large attendance and the fulness of debate, but by the reports of the proceedings that appeared in the daily newspapers. And, indeed, there are few subjects more worthy of close consideration than the physiological conditions under which the education of the young can be best carried out. The function of the school and the college has been all too much confined to the training of the mental faculties of the young, whilst their physical training has been left to chance, which is often a mischance. Happily there is in childhood and youth a buoyancy of life that leads to an amount of spontaneous exertion tending to the full development of the physical frame, and especially of the muscular powers. But where there is no proper regulation of exercise, it is apt, on the one hand, to be insufficiently carried out by the indolent or by those who have

restricted opportunities, and, on the other, to be overdriven by the eager or by those who are urged on through unhealthy emulation. In this way there are some who never have enough of exercise to ensure their full development; whilst there are others who are damaged by the too great strain that is put upon them in their games. They manage these things better in Germany. But it does not need the pressure of a paternal government to secure a wholesome system of exercise for the young. In this respect Republican America is far ahead of us, and in some of its schools and colleges a system of physical training has been set up which is almost ideally perfect.

I have on more than one occasion referred to the admirable gymnasium gifted to the College of Amherst, Connecticut, by Mr C. M. Pratt of Brooklyn, one of its old alumni. It impressed me more than anything I saw in America, and it was the one thing in all the New World that I felt disposed to covet. The ample building for compulsory and voluntary gymnastic exercise is erected in the College campus in close proximity to the various halls, class-rooms, and residences, and besides containing a great hall well stocked with all variety of apparatus for exercise of every kind, it is provided with abundant appliances of shower, sponge, and plunge baths, with hot and cold water, and dressing-rooms containing lockers for the reception of the gymnastic suit, as well as providing for its drying and airing after exercise, and for ventilation of sponge and towel. Professor Hitchcock, who has charge of the department, many years ago instituted a careful series of anthropometric observations which have been applied to all the men who enter the college. Each student when he first comes up is subjected to a very complete scrutiny, and has entered for him on his card his height, weight, strength, lung capacity, and many measurements of head, neck, trunk, and limbs, as to length and girth of various parts, as well as observations on the condition of his eyes, ears, heart, lungs, and muscles. For from his thirty years' records Dr Hitchcock has been able to construct tables which will show the student at a glance how he stands compared with the average of his own age or height. The Professor, it may be remarked in passing, is in this way accumulating a large series of valuable data, which may give more correct impressions as to the characteristics of a typical man than are to be obtained from the measurements made of soldiers, sailors, prisoners, and other groups from whom measurements have been taken usually under government requirements.

When the department was first established in Amherst, it was a question with the authorities whether compulsory physical exercise would be maintained, but experience soon taught them that properly managed physical exercises could be as well made compulsory as any other college duties, and in the afternoon or early evening, after the work of the various classes is over, the

men of each year have to go as regularly to the drill-hall as they have gone to their various classes. The idea of physical culture, as Dr Hitchcock indicates, has too often been that great muscular development is the only essential element in it; but he thinks that in order to keep the students in the best condition for work, it is demanded that the muscles be not trained to their fullest powers. He holds before him as a model, not a Hercules, but "rather the Apollo with such physical development as shall be the fittest receptacle for bodily and mental strength and grace." "Physical culture," he says in another pamphlet, "as expressed to Amherst College students, means something besides, something in addition to muscular exercise. It includes cleanliness of skin, attention to stomach and bowels, relaxation from daily mental work, freedom from certain kinds of petty discipline, but with so much requirement and restraint as will give coherence, respect, and stability to the methods of maintaining health and the men employing them. The way in which students here are called upon to secure health and its correct and normal maintenance for college requirements, is to be sure of some active, lively, and vigorous muscular exercise at stated periods; not requiring a rigid military or hardening drill of certain parts of the body, but offering them such exercises as shall, while regularly obtained, be vigorous, pleasant, recreative, and at the same time, even without a manifest consciousness of it, be calling into play their powers in active, vigorous, easy, and graceful movements. Light wooden dumbbells, weighing about one pound each, are placed in the hand, and then a series of movements are directed and timed by music, occupying in all from twenty to thirty minutes each day, which are simultaneously performed by a whole class under the lead of the captain. . . . . The young men at the close of one of these exercises, with the temperature at 60°, have usually secured moisture on the skin, are breathing fully and deeply, the blood circulates, the abdominal viscera are sufficiently stimulated, and their muscles are limber and elastic, they have gained good exercise, and the whole man has the feeling that he has worked in a physical way, and yet is not exhausted. The whole body in the loose and easy uniform, unconstrained by a rigid piece of apparatus, is given a freedom of action which cannot be acquired by the stolid march or the constraint of any fixed or many kinds of movable gymnastic apparatus; and, lastly, the students generally feel, with all, that they have had a good time. And the mental and social freedom allowed and encouraged in these exercises conduces to the rapid and healthful evaporation of superfluous animal spirits, generated by the physical and mental confinement of study."

I found it an interesting spectacle to watch a hundred young fellows carried through the various evolutions of their half-hour's drill, and one can readily understand that the effect of such systematic healthful discipline is to reduce the morbidity among the men to

quite an appreciable degree. Instead of their declining in health with their successive years of college work, it is found that their proclivity to sickness lessens. Their sickness record of a quarter of a century shows that while the percentage of sick in the first or Freshman Class is 23·5, it is only 18·3 in the fourth or Senior Class, giving a decrease of more than 5 per cent. in the morbidity from Freshman to Senior years.

I have spoken of the physical culture as it is carried out in Amherst College, because I had the good fortune to see it in happy operation there, and I then received such a strong impression of its value in developing the whole man harmoniously, that I felt that if ever I were under the necessity of sending a son from home to receive a college education, it would be to Amherst he should go, to share in the benefits of systematized intellectual and physical training. But many others of the best colleges in America have introduced systematic physical exercises as a part of the regular curriculum, and spend money freely upon buildings devoted to this purpose. Their example is being widely followed in the schools, and a demand has arisen for educated men and women to act as teachers in this department. It has been recognised that the training should be carefully graded and pursued upon a regular system, and that it is of vital importance that children and growing boys and girls should come in contact, in the gymnasium, with teachers who are as refined and competent to teach as instructors in other branches. In Brooklyn, an association exists for the advancement of Physical Education; and a Normal School for the training of teachers in this important department has been established. "Fifty years hence," they say, "it will seem strange that what was deemed by the ancients indispensable to a complete education should have waited until the nineteenth century for general recognition in our modern educational curriculum." The dilettante calisthenics or the rude rivalries of the football field, with all the ill-regulated efforts and intermediate forms of athletics that take the place of physical training among us, make one fear that the nineteenth century will be gone before British schools and colleges have wakened up to recognise the importance of aiming at the healthy upbringing of the young by making due provision for their bodily as well as their mental discipline. But it gives some ground of hope for progress, that in this Society the matter should, once at least, have been taken up and subjected to free discussion.

II. Influenza.

Another evening—the evening of March 5th, 1890—was devoted to a discussion on Influenza. Little wonder. The presence of that mysterious malady was making itself known in many ways. It raised the mortality to an unusual height,—the general mortality in our city reaching one week the unprecedentedly high rate, as Dr Littlejohn told us, of 32 per 1000. When I went to York Buildings to

register the birth of a boy on January 16th, my old friend Mr Adam greeted me with a very sad countenance, till I asked him for a birth certificate, when he brightened up, saying, "Oh, I'm glad to hear that's what you want. There have been so many deaths this week, I was afraid you were coming in for a death certificate." Apart from the increased mortality, the disease made itself uncomfortably familiar from the wide range of its attack and its tendency to recur. Its victims had many diverse forms of suffering; those who had it, even in the milder forms, were conscious often of prolonged enfeeblement; and throughout the community at large there was an unusual degree of depression of the nervous system. The discussion was opened by Dr Brakenridge in a paper which will be of permanent value as giving a very clear account of the course of the epidemic of 1890, and a careful record of its distinctive characters; and Dr Buchan, the distinguished meteorologist, added to the interest of the evening by giving us some observations as to the meteorological conditions that prevailed, and the influence of cyclonic and anti-cyclonic currents in diffusing atoms through wide areas. But I think we owe it to ourselves to confess, that in dealing with this disease we are in the face of problems still waiting their solution. It is quite in the present line of the march of Medicine to regard it as of microbial origin. We were not therefore surprised to hear that an Austrian had claimed to have discovered the influenza germ; but the confirmation we waited for has never come. No one has yet got sight of a micro-organism that can be regarded as the propagative agent in the development of influenza, much less has its geographical habitat been recognised, or the seat of its operations in the human frame, or the conditions of its multiplication and diffusion. If we do not confess our ignorance to ourselves, the laity will do it for us. Says a daily newspaper:—"We have had two winters of influenza, and now we are threatened with a third. All our doctors have had patients suffering from the malady, and a good many of them have had the disease themselves. It has attacked all ranks, ages, temperaments, and localities; and steady family physicians, whose knowledge dates back to the great attack in '48, and inexperienced young practitioners with faith in their own skill and their patients' strength, have alike sought to grapple with it. There has been every variety of patient, every variety of treatment, every variety of environment, with (we now want very much to know) what general result. Certain drugs heretofore believed in must have been proved wholly ineffectual, others lately tested for the first time may have shown themselves useful or dangerous, or doubtful. Probably the disease is on us again, and are all our experiments and experiences to go for nothing? Is the mind of the ordinary practitioner—say of the dispensary doctor in some remote country district—to remain like Locke's 'sheet of blank paper?' We want—the public, at all events, if not the profession—some

definite information, some guiding, some help. There are printed instructions in theatres of what should be done in case of fire. We want some such authentic advice in case of influenza."

Now while the demand in the close of this paragraph is well met by the admirable rules regarding the treatment laid down by Dr Brakenridge in his communication, the general drift of the remarks is justified by the scanty knowledge we have yet attained of the essence of the malady. We of this generation were hardly prepared to believe that an epidemic of influenza could have such a powerful effect on the morbidity and mortality of a community. We had been wont to use the name in rather airy fashion, sometimes to impress on some one with a slight catarrh the need of taking care; sometimes to mitigate the alarm of some others who feared they were in for graver mischief. I am old enough to remember the great debates that used to take place in this Society and city as to the efficacy or inefficacy, and worse, of bloodletting in the treatment of disease. My sympathies were with Dr Bennett when he maintained that the practice had been always futile, and the abandonment of it due to change of pathological view on the part of the profession rather than to change in the constitution of their patients. But after what we have seen these last two years, I am not so sure that the philosophic Laycock had not some good ground for maintaining that influenza epidemics which preceded the change in practice had produced an abiding effect on the human system, such as to render it less tolerant of the bleedings that had been so freely had recourse to in earlier times. However this may be, we have, in the return of this mysterious visitant in such impressive form in our generation, a challenge to grapple with the mischief. Especially it calls on the men familiar with the newer methods of observation and research, to set themselves determinedly to discover its secret, to track it to its source, and to devise the means to check its ravages. If some of our younger members will but rise to the occasion, we shall have another influenza evening that will have more definite issues and shed a clearer light upon the problems that it sets before us.

III. Tuberculin and the Cancer Microbe.

The third occasion on which we had a crowded attendance was on 3rd December 1890, when it had been announced in the billet that Dr Philip would give his "Personal Impressions of Koch's Treatment at Berlin, with Early Notes of Cases treated in the Royal Infirmary of Edinburgh," and that Dr Russell was to read a paper "On a Characteristic Organism of Cancer."

Koch's Treatment—Expectation and Disappointment.

The whole civilized world was in a ferment of expectation because it had been proclaimed that one of the ablest bacteriologists of the day had succeeded in producing a cultivation fluid that was

likely to be able to stay the progress of consumption. The very possibility that such a discovery had been made might well thrill the most indifferent and stir the dullest imagination. For to cure consumption would be to free mankind from one of the most widespread, most remorseless and deadliest of diseases. Further experience has utterly disappointed the too exalted expectations that were at first aroused, but some of our most trusted clinicians tell us they still get favourable results from the injections of tuberculin in selected cases. Koch himself, as well as Dr William Hunter and others, continue to make investigations into the composition and modes of preparation of the liquid that may lead to elimination of some of the dangers attendant on its use; and the very fact that for a moment a vista of light seemed to open in the gloom will serve to stimulate to efforts in that direction that in the end must ensure beneficent results. Meantime, we have been taught the danger of having the hand of a scientific worker forced by imperial behest, and the eagerness with which the remedy was so widely demanded by the profession, and the zeal with which it was at once put to the proof, ought to show the laity that the medical mind is not so inhospitable to new and unproved modes of treatment as it is too often alleged to be. It is to be hoped it will never deign to dabble in solutions of blue and green electricity. It must be excused from listening to the clamours of those characters that prey upon the sufferings of men, however abundant may be their alleged cures, who choose to keep their nostrum secret, or vaunt a remedy that outrages our reason. But it longs as earnestly to be able to cure, as the sick for whom it cares long to be cured; it accepts with eagerness an entirely new method of treatment when it comes with the authority of a man whose previous career Professor Gairdner could describe "as an admirable lesson in patient and cautious, as well as brilliant research and discovery;" and even when its expectation is disappointed it does not despair, but cheers the worker on to further and more fruitful effort.

A Cancer Micro-organism?

If there is any disease more universally dreaded than consumption it is cancer. To be sure it does not, like consumption, smite down remorselessly the young and promising, but those who are seized in its relentless grasp find themselves shut up in a more hopeless gloom. If consumption could be cured, or other diseases shorn of their mortality, the more numerous would become the victims of this fell disease. It was, therefore, with the greatest interest that on the same evening on which the new remedy for consumption was discussed that the Society listened to a paper by one of our esteemed Secretaries, in which he sought to demonstrate the existence in cancerous tissue of a fungus which should be characteristic of cancer, and the cause of its development. The paper was the out-

come of long and elaborate research, and though in the subsequent discussion the opinions that were expressed were mostly adverse to the acceptance of Dr Russell's conclusions—as, indeed, it is very doubtful whether cancer be of microbial origin at all—yet the communication is valuable if for nothing else than for the stimulus it gives to further investigation into the intimate nature of this variety of neoplasm. Every generation finds its crop of cancer-curers springing up; but, though sometimes remedies can be successfully applied to diseases of which the pathology is still unsettled, it is in the patient research into the exact nature of any disease that our best hope lies of learning how to check its spread, or, better still, to guard against its onset.

The Microbial Era in Medicine.

At different epochs Medicine has made rapid strides in its progress under the dominance of some prevalent idea. In the middle of this century, the idea that the unit of life was to be found in the cell prevailed, and led to the development of cellular pathology. In these later years the idea that many forms of disease are due to the action of micro-organisms prevails, and the close of the nineteenth century will be always remembered as the era of micro-biology in Medicine. The first volume has just appeared of a great *Traité de Médecine*, which is to extend to six volumes, written by a score of the younger physicians of France, and edited by Brissaud under the direction of Charcot and Bouchard. It begins with an extensive treatise by Charrin on *Pathologie Générale Infectieuse*, and thus gives micro-biology the very first place in medical study. What the issue will be of all the work that is being done under the stimulus of this idea it is hard to foretell. Whilst at this moment it looks, to us, as if the microbes were the all-important elements to be observed in diseases and their treatment, it may be that in a further stage we shall have to concern ourselves more with the chemical products that are found along with them, or with the conditions that admit of their entrance into the system. In any case it is the bounden duty of any society, or school, or city, that wants to keep in the vanguard of Medicine, to bend itself to strenuous work on the micro-biological problems of the day, and to encourage and foster by every means the talent for bacteriological research.

Edinburgh has not hung back altogether in the march. Professor Chiene, *e.g.*, has nobly striven to develop this department in the surgical laboratory of the University. But I have a misgiving that we have not done all that might have been expected of us; and it is perhaps to be regretted that our men who wish to be fully trained in this kind of research, or who have set themselves to the solution of some of its problems, have to make their way to the Pasteur Institute at Paris, or the Bacteriological Department of some German university, in order to find the proper

opportunity. We can, of course, excuse ourselves, because of the much other work we have to do, and because of the inaccessibility of a governmental purse from which the necessary provision can be obtained. But we might do better to cast about and see whether we could not do something even now that would be worthy of our good name. The medical and surgical corporations and societies might combine, and doubtless they would be backed by contributions from individual members, to promote the establishment of a Bacteriological Institute. Or the College of Physicians itself might undertake the enterprise. It would only be adding to the good service it already rendered to the profession in establishing, as it did a few years ago at the initiative of Dr Batty Tuke, the Pathological Institute in Lauriston Lane, in which already so much valuable work has been produced.

Valete!

Gentlemen, it was at one time in my mind to have spoken to you on this occasion of great men and great doings in the past of the Society. The glance at the Transactions of the past two years has led instead to this brief outlook into the future; and whilst emulation might have been stirred by study of what had been done by those who were before us in the field, it may be that a better stimulus to work will have been given by thinking of what is waiting still for us in our own day to do. We have here a happy meeting ground where the workmen from different corners gather to compare results, and it is a pleasure to reflect that in Dr Joseph Bell you will have a President who not only is a celebrated Surgeon, but who, through his long connexion with our Medical Journal, has kept himself acquainted with what is being done in many quarters, and is therefore singularly well qualified to preside over our wide-ranging deliberations.

I said that I owed you thanks, both for the honour of being called to the chair, and for the opportunities presented to me of listening to your debates. Let me thank you further for the occasion you have offered me to form and foster friendships among my fellow office-bearers. To one of a not very clubbable disposition, and consciously defective in social qualities, such occasion has been very precious; and I leave this platform at once honoured in having been your President, enlightened in many ways through your Transactions, and enriched in life with what makes life truly rich, the friendship of good men. It is, therefore, with something of real affection, as well as with heartfelt gratitude, that I say to you now—Farewell.

A vote of thanks to the President, Professor Simpson, was moved by *Dr Littlejohn*, and seconded by *Professor Struthers*.

II. Election of Office-Bearers.

The following gentlemen were elected Office-Bearers for the ensuing session:—*President*, Mr Joseph Bell; *Vice-Presidents*, Dr Brakenridge, Dr P. H. Maclaren, and Dr Claud Muirhead; *Councillors*, Dr David Menzies, Dr J. H. Balfour, Dr R. M'Kenzie Johnston, Dr R. H. Blaikie, Professor Simpson, Dr J. Batty Tuke, Dr Semple, and Mr George A. Berry; *Treasurer*, Dr Francis Troup, 1 Minto Street; *Secretaries*, Mr F. M. Caird, 21 Rutland Street, and Dr William Russell, 46 Albany Street; *Editor of Transactions*, Dr William Craig, 71 Bruntsfield Place.

III. Election of Members.

The following gentlemen were elected Ordinary Members of the Society:—J. J. Douglas, M.D., 24 Rutland Square; Robert Stirling, M.B.; J. Y. Simpson Young, M.B., C.M., 14 Ainslie Place; John Macpherson, M.D., Stirling District Asylum.

Meeting II.—December 2, 1891.

Mr Joseph Bell, *President*, *in the Chair*.

I. Exhibition of Patient.

Dr William Stewart showed a case of successful trephining for middle meningeal hæmorrhage.

II. Exhibition of Instrument.

Dr Dawson Turner showed a simple rheostat and a new form of galvanic cell.

III. Original Communications.

1. ON ECTOPIC GESTATION.

By Albert E. Morison, M.B., C.M. Ed., M.R.C.S. Eng., F.R.C.S. Ed., Hon. Visiting Medical Officer, Hartlepools Hospital.

The subject of Ectopic Gestation is one of such great importance that notes of some cases which have come under my care in general practice may be of interest. The following five cases I record, having all occurred during the last four years, show that this malady is much more common than is probably supposed, and that with increasing experience, cases may be diagnosed by the practitioner, and an otherwise certainly fatal issue averted by treatment.

Case I.—Mrs I., æt. 30. Mother of five children, eldest twelve; youngest three years of age. First seen by my brother on December 6th, 1887.

History.—She states that two years previously she had an attack of inflammation of the bowels, which, her medical adviser said, was brought on by cold during the menstrual period. This illness kept her in bed nearly three months. From that time up to two months from my first visit to her she had been well. She then menstruated normally, but had not been right since, having missed a period.

December 6th.—She complains of periodic attacks of pain in the lower part of the bowels, so severe as to confine her to bed.

On the 13th of December she had considerable hæmorrhagic discharge from the vagina, and this, together with the severe periodic pain and the probability of pregnancy, aroused the suspicion of commencing abortion. She would not allow any examination to be made, so it was impossible to form any accurate opinion. The discharge continued more or less severe every day. The pains got worse and more frequent, and her general health began to fail from sickness, loss of appetite, and pain. The temperature during the last week has been 100° F. in the morning, 101° in the evening. It was not till December 24th, however, that she would allow any examination to be made, when owing to her struggles and complaints, nothing very definite could be arrived at, although a soft, tender swelling was found in Douglas's pouch.

On December 28th, with the consent of her friends, she was induced to take chloroform, and the following condition of things was found:—

Per Vaginam.—The uterus is enlarged, cervix soft, and os patulous. Behind the uterus is the swelling discovered on previous examination to be tender on pressure. It is rounded and elastic, and extends upwards to a point midway between umbilicus and pubes. In front of it lies the uterus, the outlines of which can be clearly defined, distinct from, but closely connected with the swelling. The breasts contain colostrum, and there is a well-marked areola round the nipple. After the chloroform she was very ill with pain in the præcordial region and shortness of breath, but nothing could be found to account for this.

29th December.—Dr Murphy of Sunderland saw the patient in consultation. He made a further examination under chloroform and passed a uterine probe. His opinion confirmed that of my brother and myself that it was a case of extra-uterine gestation. The patient, however, was so ill, we all agreed that to operate would kill her, and decided to wait for a few days and see if any improvement in her general condition could be brought about. She improved somewhat by January 2nd, 1888, and on that day the following operation was performed:—

The abdomen was opened in the middle line under the spray. The tumour was found to be covered in front by adherent omentum.

It was adherent to the parietes below for the lower half, but, on dividing the omentum, was free above. Since our last examination the tumour had increased in size, and was now on a level with the umbilicus. On introducing the hand into the peritoneal cavity, the tumour was closely adherent in Douglas's pouch and round the pelvic brim, making it improbable, even not taking into consideration its size, that the cyst could be shelled out. Accordingly the cyst wall was incised after packing the abdominal cavity with sponges. A terrific hæmorrhage followed, which was almost at once arrested by pushing a sponge firmly into the wound. To this the patient undoubtedly owes her life. The placenta was situated over the front of the cyst, and this it was which had been incised. The sponge was left in the sac stitched to the parietes. The operation was finished by suturing the remainder of the abdominal wound, leaving a drainage tube in the peritoneal cavity. The patient appeared to be little the worse for the operation, and soon rallied.

January 3rd.—Dressed under the spray. The sponge was gently removed from the sac between the placenta and membranes. Bleeding at once commenced, but was arrested by plugging firmly with gauze.

4th.—Peritoneal drain removed.

5th.—Bowels moved with calomel (grs. viiss.), followed by an enema.

6th.—Plug of gauze removed and renewed. Very little discharge. No bleeding.

11th.—As the temperature since the operation has been gradually rising and the patient complains of some pain, a hypodermic needle was pushed into the sac and withdrew fluid. A director was passed along the needle, and along the groove of the director a pair of dressing forceps. By opening the forceps and withdrawing them, the opening was enlarged. About a pint and a half of blood-stained fluid, containing purulent flocculi and smelling like liquor amnii, escaped, but no fœtus. The cavity was stuffed with gauze.

25th.—On trying to remove a portion of placenta a sharp attack of hæmorrhage came on. Chloroform was given, the whole sac explored, and the fœtus (about two months) discovered and removed.

February 8th.—The sac has gradually closed up and the patient is well.

Case II.—Mrs P., æt. 37. Mother of five children, eldest 18, youngest seven years of age. First seen April 9th, 1890, complaining of severe paroxysmal pain in the abdomen of two months' duration.

History.—She has never been well since the birth of her last child seven years ago. About a year after it was born she began to menstruate, though there never was any regular period, but at intervals of from two to six weeks a hæmorrhagic discharge. The

pains of which she complains came on about two months before she consulted me.

Present State.—Patient presents a blanched appearance. She says she has never missed any periods, but has some feelings as if she were pregnant. Colostrum is found in both breasts.

Per Vaginam.—The cervix is soft and os patulous, the uterus pressed forwards by a mass, soft and fluctuating, lying in the left lateral fornix and posteriorly. Bimanually this is felt to be the size of an orange.

There is an intermittent hæmorrhagic discharge, especially marked when the pain is present.

Diagnosis.—Extra-uterine gestation.

I at once ordered the patient to bed and to be put on light diet, and morphia to be given for the relief of pain. At this time she would not consent to any operation, although the risk she ran was carefully explained to her.

May 11th.—Patient still continues in the same condition. Although she has been lying in bed for a month she has still the paroxysmal pain and hæmorrhage. The mass has increased considerably in size. She now consented to go into the Hospital under my care, and accordingly was admitted on May 12th. Her temperature varied from 98° to 99°·4.

Operation, May 17th.—Abdomen opened in the usual way. On passing the hand into the pelvis the mass described as felt per vaginam was reached. It was bound down by adhesions, and in separating these the tube was ruptured. The left tube was then ligatured and clipped away, and the peritoneal cavity washed out with warm boracic lotion. A tube was afterwards inserted and the abdomen closed. The tube was syringed every half hour until the peritoneal cavity was dry.

May 20th.—Tube removed. Bowels moved with calomel. Temperature at 9 P.M., 101°·6.

25*th.*—Patient very well indeed. Temperature, 98°·4.

31*st.*—Patient apparently quite well. I then went away from home for four days, and on my return found the patient dying. She had had sickness and diarrhœa for three days, probably owing to some indiscretion in diet, and nothing ordered had done her any good. She died of exhaustion on June 2nd, 1890.

The specimen removed showed the tube filled with coagulated, firmly adherent blood, but no trace of the fœtus could be found, and on microscopic examination no evidence of placental tissue was discernible.

CASE III.—Mrs G., æt. 34. Six children, youngest 3 years old. First seen on January 24th, 1891, complaining of obstruction of the bowels of four days' duration.

History.—Patient has been troubled with her bowels more or less for some years. About two years ago she had a similar attack

to the present one. She had been ill for four days when she sent for me, with pain in the bowels and constant sickness. The bowels had not acted for five days, and then only slightly.

On examination I found her abdomen much distended, especially in the area of the colon, and tender to touch in the left iliac fossa. There is also an ill-defined resistance in this region.

Per Rectum.—There is an obstruction high up just within reach of the finger, which feels soft and fluctuating.

Per Vaginam.—A soft, fluctuating swelling about the size of a cricket ball can be felt to the left side of the uterus in the lateral fornix. The os is soft. No discharge from uterus. Being suspicious that this was an ectopic gestation, I questioned the patient carefully as to her menstrual history, but being a stupid woman, it was difficult to obtain satisfactory information on this point. She said, however, that she had always menstruated regularly and had no uterine troubles. No colostrum was found in the breasts.

I ordered an enema and 5j. doses of magnes. sulph. every half hour till the bowels were moved. In the evening they acted, and she is much better. The sickness has somewhat abated. The swelling can still be plainly felt both per rectum and vaginam. She made satisfactory progress, still being kept strictly in bed, until February 7th, when I was sent for hurriedly at 6 P.M. She had got out of bed to stir the fire shortly before, as she was feeling so much better, when a sudden, violent pain seized her in the abdomen. She fell on the floor, and had to be lifted into bed. On my arrival the patient was extremely blanched, almost pulseless, and complaining of intense abdominal pain. On examination the flanks were dull on percussion, and it is clear that rupture has occurred, and that internal hæmorrhage is going on. I obtained the necessary instruments and assistance as soon as possible, being nearly a mile from home, and opened her abdomen. On opening the peritoneum a flood of blood and a floating fœtus escaped from the abdomen. I at once seized the left broad ligament, pulled it to the surface, transfixed, ligatured, and removed it with the placenta *in situ*. The abdomen was sponged, washed and dried, and the wound stitched in the ordinary way.

The patient lost no more blood, and made a good effort to recover, but never fairly rallied, and died on the fourth day.

The specimen removed was the left broad ligament filled with placenta and blood-clot. The fœtus was about three and a half months old.

CASE IV.—Mrs K., æt. 25. First seen October 1st, 1890, complaining of "flooding." Duration, five weeks.

History.—Patient has been married three years, and had a child fourteen months ago, which she is still suckling. She had a favourable confinement, and there is no history of subsequent trouble.

Two months prior to the commencement of the flooding she had not menstruated, but did not consider herself pregnant. During the last five weeks she has had attacks of colicky pain in the lower part of the abdomen, lasting sometimes one and a half hours, and so severe that she was compelled to go for ease on her hands and knees. The discharge has been very stringy and thick.

Present State.—She is a small, thin, neurotic woman, with anxious expression. On examining her breasts, there is a dark areola round the nipple, and some milk can be squeezed out, but as the child is still suckling these are accounted for. There is no sickness. She has some frequency of micturition and a bearing-down pain in the rectum.

Per Vaginam.—There is a soft, fluctuating swelling lying in the left posterior fornix, low in Douglas's pouch, and pressing forward the uterus against the pubes. To the left of this a firm nodule like the ovary can be felt. Bimanually the mass is about the size of a small fœtal head and fairly movable. The cervix is soft and os patulous.

I advised the patient to come into Hospital at once, but she went home, and I did not see her till the 8th of October (a week after). She then came into my consulting-room hardly able to walk and very exhausted. I at once sent her to the Hospital, and visited her two hours later. She was very breathless. Temp. 105°; pulse 160 per minute, but no blanching of lips or face. The swelling is somewhat larger than a week ago, but retains the same characters. She has had very severe diarrhœa with great tenesmus.

9*th.*—Temp. 103°·4; pulse 151. Had a restless night. Considering the condition of the patient I deemed it inadvisable to interfere, the probabilities being that she would not survive the operation. In this my brother, who saw the case with me, concurred. I ordered her peptonised milk with a tablespoonful of brandy every two hours, and powders, containing 10 grains each of soda and bismuth and $\frac{1}{8}$ grain of morphia, every four hours.

11*th.*—Patient very prostrate, gradually getting weaker; the diarrhœa continues. I examined her again and found the swelling larger. As there seems a possibility that the continued high temperature is due to suppuration in the sac, I explored the swelling through Douglas's pouch with an aspirator needle under strictest antiseptic precautions, but only got a few drops of *old* blood.

14*th.*—Patient very weak. The diarrhœa is persistent, the abdomen tympanitic, but not tender. She is dull and heavy, and only answers questions when put to her in a loud voice. Pulse 140, small and feeble. Ordered 5℥ doses of Tinct. digitalis every two hours.

16*th.*—Patient quite violent, wants to get out of bed, and imagines she sees objects. Temp. 104°·6. Tongue dry and cracking. Diarrhœa still persistent, and she has lost control over both sphincters.

17*th*.—Lies on back quite unconscious, cannot be roused, taking almost no nourishment. Temp. 104°·6; pulse uncountable.

21*st*.—Died early this morning, the high temperature continuing and the diarrhœa persisting to the last.

Post-mortem Examination, 30 hours after death.—The pelvis was removed entire from the body and showed the following:—There is a small pyosalpinx on the left side. The right tube is greatly distended with coagulated blood in layers. The tube has ruptured into the broad ligament, and at least a quart of blood forced its way down between the peritoneum and the pelvic fascia, stripping off the peritoneum from the floor of the pelvis (extra-peritoneal hæmatocele). The right ovary is lying posteriorly. The uterus and bladder are jammed against the pubes and the rectum against the sacrum by the mass of extravasated fluid and clotted blood. The high temperature and the diarrhœa cannot, to my mind, be accounted for in any other way than by supposing it to be due to the absorption of pyrogenous material from the blood extravasated into the pelvis, as no other disease was present, and death resulted from the hæmorrhage.

Case V.—Mrs L., æt. 34. First seen September 9th, 1891, complaining of a "dragging pain in the right side and constant and excessive loss." Duration, three months.

History.—She is the mother of seven children, the youngest being sixteen months old. She always had good times at her confinements. The last was very quick, but she recovered slowly. Although she got up on the 10th day she never felt well, having a constant pain in her vagina. The child was weaned at nine months. She menstruated six weeks after this (*i.e.* four and a half months ago) and continued regular for three months. After this, menstruation became irregular, commencing a week after, stopping for two or three days, and continuing more or less up to the time she consulted me. When this irregularity began she had a very severe loss, so that her neighbours in attendance suggested a miscarriage. Six weeks ago, when cleaning the grate, she was seized in the rectum with a severe bearing-down pain, "as if something must come through her." She felt very faint and had to be put to bed. This pain left her very weak, and continued off and on up to three weeks ago. Since then it has not been violent, but an aching, gnawing sensation remains. She has had more or less hæmorrhagic discharge ever since. Bowels are regular; no trouble with micturition.

Present State.—Patient well nourished, rather anæmic, with a worn-out, anxious expression. She complains of a dragging pain in the right side, and morning sickness. There is colostrum in both breasts.

The abdomen is very flaccid. On palpation the right kidney is felt to be movable; nothing else abnormal can be detected.

Per Vaginam.—The uterus is jammed forward. The cervix is

soft, os patulous. Behind and to the left of the uterus is a semi-fluctuating mass about the size of a fist, immovable and slightly tender. Along its under surface is felt a band (tube ?) and a softer and smaller swelling. Bimanually it can be indistinctly felt.

The patient was admitted into my private hospital on September 14th, and on the 15th I performed the following operation :—

The usual abdominal incision was made. On entering the peritoneal cavity the omentum was found adherent to the abdominal wall, and the intestines in the pelvis were matted together. On getting down to Douglas's pouch a mass of adhesions was encountered, and on breaking through these my hand came on a cavity filled with blood-clot. The right tube was twisted over to the left, distended, and adherent. On breaking down the adhesions it was pulled to the surface, and found to have ruptured. The corresponding broad ligament was transfixed, tied with a figure-of-8 ligature, the diseased appendage clamped and clipped off. The left tube and ovary being healthy were left.

The abdomen was well washed out with sterilized normal saline solution at 110° F. During this process many coils of intestine protruded, and were with difficulty replaced. A drainage tube was left in Douglas's pouch. The patient was put back to bed very cold, evidently from the manipulation required to return the gut, but her pulse never varied nor failed.

16*th*.—Patient had a good night; very little discharge from tube. Temp. 98°·6 ; pulse 100.

17*th*.—Good deal of distension ; seidlitz powder, and turpentine enema. Temp. 99°·4; pulse 112. One oz. from tube.

19*th*.—Patient very feverish and wakeful all day. Temp. 102° ; pulse 125. A good deal of watery fluid from tube ; abdomen very tender, no distension ; ice applied to head.

20*th*.—Patient very drowsy, sleeping at intervals, breathes heavily. Temp. 102°·6 ; pulse 126. Bowels well moved ; very little from tube, which was removed. Ordered grs. iij. of calomel every two hours, and to be sponged with tepid water; patient menstruating.

21*st*.—Patient had a good day, free from pain. Temp. 99°·8 ; pulse 100. After this patient had no drawback, and was able to go home on the 17th day.

Remarks.—Of the five cases, four were submitted to operation, the fifth was too ill to allow of any operative interference.

Of the four patients operated upon, three recovered and one died. Amongst the recoveries I include Case II., who died before dismissal from the Hospital on the 21st day after operation, but from causes entirely independent of the operation. In all three patients who recovered the intra-peritoneal bleeding was limited by adhesions, the result of previous pelvic peritonitis. In the patient who died, bleeding was not thus limited, hence the sudden and fatal calamity.

In three cases the diagnosis was confirmed by the discovery of a

fœtus, but in the other two no trace of fœtus or placental tissue could be found, after most careful search, in one after operation, in the other after post-mortem examination. Am I then justified, in the absence of such proof, to include these two cases under the heading of ectopic gestation? Admitting the possibility of error, I feel strongly that the clinical evidence is all in favour of this course—for

α. The history of the cases is very suggestive.

β. The conditions found at operation, whether a fœtus is found or no, are too similar to escape notice.

γ. The termination in death seems equally certain in both.

Then it may be asked, What other condition could produce spontaneous bleeding into a tube, rupture of the tube, and fatal hæmatocele? I can imagine no other without soaring into the realms of fancy.

The objections are surely easily disposed of. Is it remarkable that a fœtus of from six weeks to three months old should be lost in the peritoneal cavity? Does not every work on abdominal surgery particularly warn the operator of the danger of leaving sponges, even large ones, in the abdomen? Is it not true that dead animal tissues are quickly consumed in the peritoneum? Considering these facts, it appears to me remarkable, rather, that so many proofs of this sort can be produced. Anyone accustomed to histological work will appreciate the difficulty of producing satisfactory proof in the form of placental tissue from the débris dug out at such an operation. If it is found, the finder is fortunate and the diagnosis certain; if not, no one is to blame, and the diagnosis is not negatived by the misfortune.

In Case III., the symptoms were those of intestinal obstruction, and for this alone advice was sought. No clue was given by the woman, though she was frequently and carefully questioned, which would have helped to confirm my suspicions that an ectopic gestation existed, and it was only the intra-peritoneal rupture that rendered it sufficiently certain to justify immediate operation. Had the patient not been speedily relieved of her intestinal symptoms, I should certainly have opened her abdomen, and with every possibility of saving her life.

Curiously enough the only two cases in which portions of membrane resembling decidua could be demonstrated in the uterine discharges were the two (Nos. 2 and 4) in which no trace of fœtus or fœtal structures was discovered, though in all cases the discharges were carefully examined by me whilst the patients were under my observation. This raises the question of the value of this sign.

In three of the cases the patients had, as in Case I., a previous attack of peritonitis; or, as in Cases II. and V., had never felt well since the last confinement.

Four of the cases had two pronounced symptoms, viz.—(1)

recurring uterine hæmorrhages, and (2), severe attacks of colicky pain in the lower part of the abdomen. Rectal tenesmus occurred sufficiently often to make the symptoms worthy of attention. The most characteristic symptoms and signs met with were—

1. A history of previous pelvic mischief.
2. Many of the symptoms of pregnancy, changes in the breasts, vagina, os, and cervix.
3. Recurrent attacks of hæmorrhage preceded by painful spasms in the lower abdomen.
4. The presence of a tender, elastic swelling behind or to one side of the uterus, which increased rapidly in size.

All the cases were under observation from one to six weeks, and in all this progressive enlargement was noted.

To sum up, where symptoms and signs such as are described exist, there are sufficient grounds for the diagnosis of ectopic gestation, and the abdomen should be opened without delay.

Dr Berry Hart congratulated Dr Morison on his most valuable paper. To the diagnosis and treatment no exception could be taken, although extra-uterine gestation was one of the most difficult subjects in obstetrics. Dr Morison's first case was evidently an extra-peritoneal gestation, and considering the difficulty of the operation the good result was remarkable, and spoke highly for the operator's skill and daring. He (Dr Hart) was glad Dr Morison was so sound in his treatment; there was nothing so good in selected cases as abdominal section. The paper was a most valuable practical contribution.

Dr Haultain begged to thank Dr Morison for his report of such an interesting series of cases. In Case 1, which showed all the signs of an extra-peritoneal hæmatocele, he would have been inclined to have let the patient alone, as in these conditions the effused blood as a rule became absorbed, and the cases tended towards spontaneous resolution and cure. If secondary suppuration did occur, the cavity could be opened, and washed out either through the abdomen or vagina, the risk of hæmorrhage being now diminished. He was surprised at the few cases in the series in which free intra-peritoneal hæmorrhage was met with, and thought Dr Morison was to be congratulated on its absence. In one case upon which he had had the pleasure of assisting Dr Halliday Croom in operating, rupture of the tube had not occurred; but from separation of the ovum from the tube-wall extensive hæmorrhage had taken place through the distal end of the tube, but fortunately in this case the tube opened freely into a separate sac in which the ovary lay flattened out. This sac formed a tumour bigger than a large orange, and was distended with dark fluid blood. It evidently was an example of that developmental condition described by Bland Sutton, in which the ovary, like the testes, has a complete peritoneal investment surrounding it, into which the Fallopian

tube enters, with its fimbriæ hanging perfectly free, thus removing the possibility of an inflammatory formation. He again thanked Dr Morison for his valuable paper, and looked forward to its more thorough study when it appeared in print.

Dr J. W. Ballantyne had listened with much interest to the recital of cases which Dr Morison had given. He had heard some years ago of Dr Morison's first case, and was now glad to learn that the patient had entirely recovered, a result which he thought was, under the circumstances, most satisfactory and encouraging. Such a successful termination indicated that, even in cases where the gestation was probably extra-peritoneal, laparotomy might be performed with a reasonable hope of cure. About a year ago he had seen a case in consultation which demonstrated how tedious and dangerous might be the progress of events when an ectopic extra-peritoneal pregnancy was either not operated upon or only opened per vaginam. The patient to whom he referred had for nearly a year and a half been in imminent danger, had been quite confined to bed, and had been passing fœtal débris through the vagina; she had ultimately recovered, but only after very prolonged suffering.

Dr Morison, in reply to Dr Hart, said he looked on Case 1 as being extra-peritoneal. That while admitting the importance of being able to demonstrate the presence of placental tissue, still he thought that even those cases where it was not found were cases of ectopic gestation. In reply to Dr Haultain, he thought Case 1 was undoubtedly the case in which operative interference was justifiable, and that he would consider it bad surgery to delay until suppuration had taken place in the sac, and then open per vaginam. The risks of prolonged suppuration and septicæmia were too great. The reason the hæmorrhage in the cases recorded (except 3) was limited was that adhesions had formed over the pelvic brim.

2. ON CERVICAL RIBS, WITH EXAMPLE IN LIVING SUBJECT.

By David Wallace, M.B., C.M. Ed., F.R.C.S. Ed., M.R.C.S. Eng.

The occurrence of cervical ribs in man is, I think, of sufficient rarity to justify me in laying before this Society a short note upon them from an anatomical point of view, before showing a patient who presents the peculiarity. Apart from variations in shape, anatomically three variations in ribs are specially referred to by anatomists, viz.,—(1) *Rudimentary*, (2) *Bicipital*, and (3) *Supernumerary*. The rudimentary and bicipital varieties are of less interest to the surgeon than the supernumerary; so I may dismiss them by merely stating that the highest and lowest ribs are apt to occur in an imperfectly formed condition, sometimes the first and

sometimes the twelfth being so small as to represent, as said by Professor Struthers,[1] the rib shrinking into the transverse process of the dorsal vertebra, just as the supernumerary cervical rib may be said to represent the anterior tubercle of the transverse process expanded into a rib. Professor Turner,[2] writing of the bicipital rib, says,—"The anatomical peculiarity is not due to a bifurcation of the shaft of a single rib at its vertebral end into two heads, but to the fusion of what should have been the shafts of two distinct ribs into a common body. It invariably occurs at the apex of the thorax."

Supernumerary ribs occur as an addition on the seventh cervical or first lumbar vertebræ, but undoubtedly they are much more frequently in relation to the seventh cervical vertebra than the first lumbar. Professor Struthers has described, in the *Journal of Anatomy and Physiology*,[3] a specimen of a lumbar pair of ribs constituting the lowest pair of thirteen pairs of ribs. The occurrence of supernumerary ribs had been observed by Galen, Fallopius, and others, but they, as Dr Dymock[4] writes, have forgotten to favour us with the details, and we are ignorant of the region in which they were met with. M. Hunauld,[5] 150 years ago, described nearly all the forms of cervical ribs which have in more recent years been noted; and further, as pointed out by Dr Dymock, he was the first to accurately describe their developmental formation.

Professor Grüber,[6] in 1869, published the results of an elaborate investigation into the question, and in a short statistical table shows that from the time of Hunauld (1740) down to that year, 76 examples had been recorded of cervical ribs occurring in 45 individuals. Since then numerous other cases have been put on record, notably by Sir William Turner[7] (7), Professor Struthers[8] (10), and Professor Shepherd[9] (4).

Professor Struthers, after reference to the variations in the shape and length of the seventh cervical transverse process, says that he believes the occurrence of cervical ribs is more frequent than supposed, but that in many instances they are merely rudimentary, and the part not being closely dissected, they escape notice.

The majority of specimens described have been discovered in the dissecting-room or after maceration; and although varying much in size, and on this account in relationship to other structures, it is found that they can be conveniently grouped under two divisions —1*st*, Those which are short and possess merely a head, neck, and

[1] *Vide Anatomical and Physiological Observations*, by Professor Struthers 1854.
[2] *Vide Journal of Anat. and Phys.*, vol. xvii.
[3] *Ibid.*, 1875.
[4] *Vide Edinburgh Medical and Surgical Journal*, 1833.
[5] *Vide Mémoires de l'Academie Royal des Sciences*, 1740.
[6] Grüber, *Memoirs of the Imperial Academy of St Petersburgh*, 1869.
[7] *Op. cit.*
[8] *Op. cit.*
[9] *American Medical Journal*, 1883.

tubercle—the short process beyond not being worthy the name of shaft; and, 2*nd*, Those which, in addition to the above, are so developed that they possess a shaft, and it may be extended so far as to articulate by cartilage with the sternum. For a long while no such perfect cervical rib was found, and Sir George Humphrey, in his book *The Skeleton*, published 1858, states that this does not in any case he knows of happen. Professor Struthers mentions a case where on the left side the shaft was prolonged to the sternum, but the cervical rib in that case joined the cartilage of the thoracic rib nearly an inch from the sternal margin. Through the kindness of Sir William Turner, I am able, however, to show you a perfect cervical rib in position, and it articulates directly with the sternum by a broad strong cartilage.[1]

The true morphology was first pointed out by Hunauld. It is, Quain states, "very generally admitted that the part in front of the vertebrarterial foramen of the cervical vertebra corresponds in series to the first part of a rib;" and thus the anterior tubercle of the transverse process of the seventh cervical vertebra may, remaining separate from the vertebra, shoot beyond its ordinary dimensions and run parallel, or very nearly parallel, with the first thoracic rib, and end half-way to the sternum as a floating or free rib resembling the asternal ribs of birds. It may, however, join the first rib, or, as in the specimen above referred to, pass forwards and articulate directly with the sternum. This excessive ossification, though usually restricted to the seventh cervical, may be seen in the sixth also, as in a preparation described by Struthers "as showing the rudiments of cervical ribs on the sixth and seventh vertebræ." This was in a male subject aged four years.

The relationship between a cervical rib and the soft parts in great part depends on the length of the rib, and it has been pointed out that if the rib be short, consisting of head, neck, and tubercle, the anterior scalene muscle is, as ordinarily, attached to the scalene tubercle on the first thoracic rib, while the subclavian artery lies anteriorly to the cervical rib. On the other hand, if the cervical rib be longer and join the cartilage of the first thoracic rib, the anterior scalene muscle may be attached to it, and the subclavian artery groove its upper surface posteriorly to the muscle. No definite rule, based on the length of the rib, can, however, be laid down for these relationships. The brachial nerves may also pass over the cervical rib; and in a specimen shown by Dr Symington to this Society in 1883, the eighth cervical and first dorsal nerves passed over the rib, the others passing under it over

[1] Unfortunately in this specimen, in which the spinal column and ribs are in position, there is an abnormality in the vertebræ—one, possibly a cervical, being absent. Professor Turner explains the peculiarity by the view that the last lumbar vertebra is anchylosed to the sacrum.—*Vide op. cit.*

the first thoracic. Dr Symington's specimen was a short rib ending in a well-marked tubercle, but attached to the thoracic rib by a strong bony process just at the scalene tubercle.[1]

A few months ago I observed, when examining a patient's chest at the Royal Public Dispensary, that there was present a prominence above the right clavicle, which, on palpation, was hard and bony. On examination of the prominence, I concluded that it was a supernumerary rib. I showed the case to Professor Chiene and Sir William Turner; and as they corroborated me in the view I held, I thought it might interest the members of the Society to see the patient.

The patient, Hugh M., æt. 60, is a labourer, who states that he never received any injury to the right shoulder, and has not previously had his attention attracted to a swelling situated in the right supra-clavicular region. He has no pain nor discomfort in the right arm.

Present State.—Comparison of the supra-clavicular regions reveals the presence of a prominence on the right side, which is situated opposite the middle of the clavicle, with its centre 4 inches from the mesial plane of the body, and its highest point $1\frac{3}{4}$ inches above the clavicle. The prominence is immediately under the skin, which moves freely over it. It is a rounded, hard, bone-like swelling, the outline of which can be felt at the upper, inner, and inferior margins. Externally, however, it is continuous with a hard flat surface which passes outwards, upwards, and backwards until it is lost under the anterior edge of the trapezius muscle. On drawing the arm down, the prominence, which is about the size of a walnut, does not move, but can be more distinctly felt; while, on the contrary, if the arm be pushed upwards, the prominence is hidden by the clavicle. To the most internal part of the prominence a band is attached, but whether this is muscular or fascial it is difficult to say.

Subclavian Artery. — The subclavian artery is felt pulsating most distinctly immediately in front of the extremity of the cervical rib, *i.e.*, it lies exactly in the angle formed by the clavicle and anterior end of the rib. No pulsation can be detected externally to the point of rib, and the artery seems to dip under the point of the rib, and then under the clavicle. Pulsation is felt as high as a line drawn horizontally through the point of the rib. Probably this portion of the artery is the third part of the subclavian.

The *anterior scalene* cannot be felt to be inserted into the cervical rib.

[1] At this point in the paper Dr Wallace showed specimens from the Anatomical Museum of the University. These specimens, which he was enabled to show by the kindness of Sir William Turner, Curator of the Museum, were chiefly a series collected by Dr Robert Knox, which was described and figured by that anatomist in the *London Medical Gazette* of 1843 in a series of papers on "Transcendental Anatomy."

Immediately above the extremity of the supernumerary rib a smaller bony prominence is detected, which seems to curve backwards in a direction parallel to the cervical rib, and suggests the possible presence of a second supernumerary rib from the sixth cervical vertebra.

On the left side there is nothing similar to *the cervical* rib, but a process similar to the second process is felt, and this has precisely the same relations.

Anatomists having drawn attention to the presence of cervical ribs, clinical observers have from time to time had their attention directed to the peculiarity during life, and numerous cases are recorded where mistakes in diagnosis have been made. Professor Turner refers to this possibility in his paper in the *Journal of Anat. and Phys.*, 1870, vol. iv., and describes, among others, a case he saw in Sir J. Paget's Wards at St Bartholomew's Hospital, and quotes as follows from a letter he received from Sir J. Paget:—

"In each case the imitation of aneurism was close enough to deceive an unwary surgeon; but to one who examines closely, and has in his mind what the case may be, the mistake seems scarcely possible so long as the artery is healthy. I can well believe, however, that great difficulty of diagnosis would exist in any case in which the unusual arrangement of parts is combined with a morbid state of the artery, especially with that state in which arteries, not evidently diseased in texture, have more than natural pulsation. This state is common in the abdominal aorta, and I have seen it two or three times in subclavian arteries and in carotids."

Sir G. Humphrey and Professors Struthers and Shepherd[1] also refer to possible mistakes in diagnosis; and I think, from the specimen No. 1, which I have shown to-night, there can be no doubt but that the possibility of an aneurism would occur to one if he had been called on to examine the case during life. While in the patient I have had the honour to show you the resemblance to an exostosis is very close, and the mistake might easily have been made.

In those cases where the rib is short, and ends in a tubercle or knob, but in which the subclavian artery does not pass superficially to it, the resemblance to an exostosis is a marked one; while, on the other hand, when the artery passes over the cervical rib, it is so high, and the pulsation is so superficial, the mistaken diagnosis of an aneurismal swelling is highly probable.

The presence of a cervical rib ordinarily is not troublesome to the possessor, but where the artery is high, an injury to the vessel might very readily occur.

Mr Caird stated that he had seen what he believed to be two cases of cervical rib,—one in which an aneurism was simulated on

[1] Shepherd, *American Medical Journal*, 1883.

the left side of the root of the neck in a woman; and a second, with which a neuritis and obliteration of the right radial pulse was associated.

3. CYSTS OF THE TONSILS, NOSE, LARYNX, AND EAR.

By P. M'Bride, M.D., F.R.C.P. Ed., F.R.S.E., Aural Surgeon and Laryngologist, Royal Infirmary, Edinburgh, and Lecturer on Diseases of Ear and Throat in the Edinburgh School of Medicine.

It is no part of my object to bring before the reader an exhaustive account of the occurrence of cysts in these parts. Indeed, with but slight digression, I shall confine myself mainly to a short statement of facts which have come under my own observation.

A careful perusal of English, American, and Continental works on throat diseases will reveal the fact that cysts of the tonsils are rarely if ever mentioned. It is, however, quite common to meet with small whitish-yellow areas, both on the glands themselves and in their immediate neighbourhood. I do not refer now to the cheesy accumulations which are often found partially extruded from a mucous follicle or crypt, and which are easily pressed out as cheesy ill-smelling plugs. In the class of cases to which allusion is intended, the deposit is covered by a layer of mucous membrane, and the appearance presented is, owing to this fact, somewhat similar to a pointing pustule. Careful inspection reveals, also, that the yellowish area bears upon its surface a fine vascular network. After puncturing and exercising a small amount of pressure on the adjacent parts, a somewhat cheesy nodule of white colour becomes extruded, differing only in the absence of fœtor from the cheesy masses above referred to. The condition just described is practically a cyst due to retention of exuded matter and desquamated epithelium, either within a gland or a crypt. I have, however, met with very definite *retention cysts in the tonsils* on two occasions. In both cases the patients were females. In neither was pain complained of, but only discomfort. On the affected tonsil was seen a yellowish-white area of considerable size over which small vessels ramified. Incision gave exit in each case to about a drachm of cream-like fluid resembling pus, and without any odour or even bad taste. In my first case I immediately excised a considerable portion of the outer wall, while in my second I contented myself with evacuating the retained secretion, and telling the patient to return if any renewal of discomfort ensued. In the former I know that a perfect cure resulted; while, as the latter has not returned with any complaint, I presume that a similar result followed simple incision in her case.

These cases have appeared to me of sufficient interest to record, because both from my own experience and from my reading I

believe such instances to be extremely rare. The etiology of such cysts can be very simply explained. Although in the human subject it is doubtful whether glands usually discharge into the crypts, yet we must remember that within them there is constantly occurring a destruction of the epithelium and an exudation of leucocytes presumably associated with more or less fluid. If, then, owing to previous changes of an inflammatory nature, a crypt has been changed into a closed cavity, we may expect it to become filled with creamy contents, such as were present in the two cases just described.

In the naso-pharynx cystic formations may occur in exactly the same manner. Tornwaldt ascribed them to retention of fluid in what he described as the bursa pharyngea. It is now, however, believed that this cavity, when present, is merely a result of inflammatory changes in one of the furrows of the pharyngeal tonsil.

Cysts of the nose are by no means very common. Hydatid cysts and the dermoid variety have both been described, and, through the kindness of Mr Caird, I was enabled to see a case in which one of the latter had been spontaneously extruded from the nose. The nasal chambers, afterwards examined, gave no evidence of either the seat of attachment or mode of origin.

Johnson, Lefferts, and Horsley have described cystic growths arising from the turbinated bodies. I do not know whether these were merely instances of degeneration of mucous polypi or primary cysts. In Horsley's case the growth was thought to be attached to the inferior turbinated body, so that in all probability its etiology could not be explained on this hypothesis. Spencer Watson has met with a case in which a large polypus was entirely converted into a multilocular cyst. I have also seen a large mucous growth having all the external appearances of a polypus, but which consisted merely of a cyst wall with fluid contents. Voltolini seems to have regarded this cystic degeneration of nasal polypi as not of any very great rarity, but in this respect his experience has probably been unique.

A form of cyst which I have met with on two occasions has the following characteristics. On inspecting the nares anteriorly a swelling is seen projecting below the inferior turbinated body, and on pressing upon this with the probe fluctuation is detected. In both of my cases the finger applied just outside of the corresponding ala, where a slight fulness was visible, could detect fluctuation, and inspection of the nasal projection during the process showed movement of the bulged mucosa. Fluctuation could also be elicited by inserting one finger under the upper lip while pressing with the other over the full area at the side of the nose. It will thus be evident that in each of the two cases (for they were exactly like each other) examination revealed a fluid-containing cavity bounded below by the labio-gingival fold,

laterally by the cheek just outside of the nasal cartilage, and internally by the mucous membrane lining the outer wall of the inferior meatus at its anterior part. On an exploratory puncture these cysts yielded a considerable quantity of a straw-coloured and apparently serous fluid. Unfortunately the amount was not measured, nor was the liquid examined either chemically or microscopically. As both these patients were seen in an out-patient department I have no exact notes of the symptoms which actually directed attention to the nose. In one of them, however, there was considerable pain in this part, which was eventually relieved by the removal of the cyst. The treatment adopted varied in the two cases. In one a cure was probably attained by puncturing from the inferior meatus of the nose, which was twice repeated as the cyst refilled after the first escape of fluid. I say probably, because after the second repetition of this little operation the woman did not return to hospital, and was therefore, I presume, relieved. The second case, however, proved much more obstinate. Opening with the electric cautery, with subsequent introduction of the incandescent point into the cavity succeeding in exciting suppuration, after which I introduced a drainage tube, but the latter had to be abandoned owing to the impossibility of keeping it in position. Eventually I requested my colleague, Mr Duncan, to dissect out the cyst wall by reflecting the upper lip, which was done, and resulted in a cure.

The etiology of the cysts I have just described is not by any means clear. I regret extremely that owing to the casual way in which the cases were first met with, *i.e.*, among a crowd of others in an out-patient department, it was impossible to make accurate observations upon the microscopic and chemical characters of the fluid contents of the cysts. So far as could be made out, any connexion with the superior maxilla or teeth could be definitely excluded, so that in considering the etiology of the cysts we are thrown back upon an hypothesis which makes a gland responsible for their production. Whether retention occurred in one of the labial or in one of the numerous nasal glandules I do not profess to say.

Another form of nasal cyst is of considerable interest and great rarity. It is characterised by the presence of an air-containing cavity in the middle turbinated bone, which enlarges to such an extent as to cause symptoms of nasal obstruction, associated or not with headache. Cases of this kind are generally, if indeed not always, met with in female patients; and examples have been described by Glasmacher, Schäffer, Heymann, Schmiegelow, Greville Macdonald, and myself. In my own case, examination of the nostrils — for the condition was bilateral — revealed rounded tumour-like bodies, apparently incorporated with the middle turbinateds, but reaching almost to the floor of the nose. On touching these with a probe they were felt to be of osseous con-

sistence, but yet firm pressure seemed to be followed by a sensible yielding. By means of a common awl I perforated one of the growths, and entered an air-containing cavity. This proceeding I repeated on the other nostril. The subsequent treatment, however, differed on the two sides, for on one I removed a portion of the cyst wall, while on the other I contented myself by squeezing the walls into a shape more consistent with the normal size of the parts. Subsequently, however, the latter again returned to its original shape, and the patient disappeared from treatment, dreading further operation. The etiology of these tumours is in one sense quite clear, for in the middle turbinated body air-containing spaces may and do exist. Why these should in certain cases enlarge, or whether in patients in whom this bladder-like enlargement exists the abnormality has dated from birth, are questions which await further solution, although Heymann in one case was able to observe that a gradual expansion of the air cavity occurred.

In connexion with cystic enlargement of the middle turbinated body, it may be of interest to note that Bayer has described a case in which this bone was converted into a multilocular cyst containing a sticky yellow fluid, that Major has made a somewhat similar observation (the only difference being that in his case the cyst appears to have been attached to and not incorporated with the middle turbinated bone), while B. Fraenkel has placed on record an instance of cystic enlargement associated with empyema of the contained cavity.

Cystic growths in the larynx have been observed more frequently than any of the forms just described. Such neoplasms have been met with arising from the epiglottis, the ary-epiglottic folds, posterior wall of the larynx, ventricle of Morgagni, and even from the vocal cords. Yet the condition is sufficiently uncommon to warrant a brief account of a case which derives additional interest from the age of the patient.

In the autumn of 1889 a woman, aged 74, consulted me on account of loss of voice. On examining the larynx, I found a small neoplasm about as large as a pea attached to the right side and situated near the anterior commissure. Considering the age of the patient, and taking into account the absence of all other symptoms, no operative treatment was advised. It was therefore resolved merely to keep the case under observation. On November 12th, however, the tumour was found to have grown to the size of a small hazel nut, and although there was but little dyspnœa, it was evident that a corresponding further increase in size must lead to embarrassed respiration. The tumour appeared as of globular shape, grayish translucent colour, and over its surface coursed dilated vessels, the whole indicating tension. As this was the first case of laryngeal cyst I had encountered, I failed to make a diagnosis before operating. At the same time, I believe that were

I again to meet with an example of this rare affection, the appearance of the tumour alone would be sufficient to indicate its character. If any doubt existed, illumination of the larynx by transmitted light would probably dispel it. Having, then, recognised that I had an innocent tumour of more than usually rapid growth to deal with, I sprayed the larynx with a 10 per cent. solution of cocaine, and succeeded in grasping the tumour on the first introduction of Morell Mackenzie's forceps. On pulling it outwards the mass collapsed, with the escape of some fluid, and a piece of membrane nearly as large as a sixpence came away between the blades of the forceps. The patient's voice was immediately restored, and on laryngoscopic examination it was found that the seat of attachment had been the anterior part of the right ventricle of Morgagni. In the course of last year I saw the patient, who had a perfectly sound larynx.

No doubt this was an instance of a retention swelling, due to enlargement of one of the numerous glands situated about the ventricle.

Cysts in and around the auricle have been described by various observers. Those most commonly met with seem to have been either sebaceous tumours or a variety of perichondritis giving rise to the effusion of serum between cartilage and perichondrium, classified by Hartmann among cystic formations. Whether the last-named are not usually a modification of the well-known blood cysts or othæmatomata seems an open question. As pointed out by Urbantschitsch, in cases where a fistula auris congenita is present, the external orifice may become blocked and give rise to a retention tumour.

Cysts in the external auditory canal are of extreme rarity, and beyond a very general reference to the possibility of their occurrence in the last edition of Gruber's work, and a quotation from the same by Steinbrügge, the matter does not seem to be alluded to in otological literature. I therefore make no apology for describing the following case:—

A middle-aged woman presented herself at the Infirmary with a history of sudden deafness in the left ear of a fortnight's duration. On testing the hearing, the watch was not heard close to the left ear. On examination the deeper portion of the meatus was seen to be entirely filled by a grayish tumour of tense appearance, and presenting on its surface ramifying vessels. On touching it with the probe it was found to be insensitive, and seemed to fluctuate very distinctly. There was no history of pain or traumatism. These facts enabled me to suggest that the growth was a cyst arising from the upper and anterior part of the osseous meatus. I accordingly punctured the swelling and evacuated an apparently serous fluid. On examination afterwards the walls were seen to be entirely collapsed, and the seat of origin from the anterior superior part of the osseous meatus was manifest. Immediately

afterwards the watch was heard clearly, and a whisper at 12 feet, so that the patient declared she heard quite well. In this, however, she was mistaken, as shown by the amount of the hearing power and by the evidence of chronic changes in the drum membrane. She was kept under observation for several days, but there was no tendency to recurrence.

The chief interest in this case centres in the fact that a cyst arose apparently spontaneously in a situation in which it is usual for histologists to deny the existence of gland structures. Manifestly many hypotheses are conceivable, but it seems useless to speculate upon probabilities where no proof is attainable.

Dr R. M'Kenzie Johnston wished to express his thanks to Dr M'Bride for his paper, which dealt with a subject of great interest. Little if any reference to these diseases would be found in any of the best text-books, and so his paper was of special interest and value. He was inclined to think, himself, that the supposed rarity, specially of cysts in the tonsils, was due to the slight amount of discomfort often produced by them, and to a tendency to spontaneous rupture and disappearance. He observed that Dr M'Bride made hardly any reference to the pathology of these growths, and he wished to know whether that was due to his not being satisfied with the theories that had been advanced. In the formation of nasal cysts M'Donald had suggested that hypertrophy was followed by fissuring, which finally caused union of the incurved surface, producing invagination of a portion of the secreting mucous membrane.

Professor Struthers asked Dr M'Bride whether he accepted as accurate the common statement that glands are not present in the osseous stage of the meatus.

Dr Horsley thought that, given the possibility of extended examination, the diagnosis between true cysts and polypi which had undergone cystic degeneration might be assured. He gave an account of a cyst occurring in the course of a case of hypertrophic rhinitis, and suggested that nasal cysts might very frequently exist in such cases without causing a disturbance in excess of that due to the general hypertrophy, and consequently be overlooked. Another cause of their being overlooked might possibly be the spontaneous evacuation of the cysts.

Dr M'Bride begged to thank the Society for the kind way in which his paper had been received. In reply to Dr Johnston, he begged to say that he had as far as possible in his paper considered the pathology, but in some of the cases it was really impossible to give a satisfactory account of the etiology, which must be only conjectural. In reply to Professor Struthers, he believed that the observations denying glands in the anterior part of the osseous meatus was accurate, although he had not made any personal observations on the subject. In conclusion, he begged to remind Dr

Horsley that it was impossible to draw a hard and fast line between cystic mucous polypi and true cysts.

The following is the description of the case of successful trephining for middle meningeal hæmorrhage at the point of *contre coup* of the patient exhibited to the Society by William Stewart, M.D., Surgeon to Leith Hospital (see page 14) :—

T. J., aged 46, in the employment of the Dock Commission, fell into the docks at eleven o'clock on the evening of the 30th September of this year, striking a wooden pile in his fall. He was soon pulled out of the water, and was quite conscious, and began to run with the rope of a ship which was leaving the docks, but was urged to go home. He proceeded to do so; but on his arrival at the dock gates, twenty minutes after his fall, and while telling the policeman on duty what had happened, he groaned, fell down, became unconscious, and was convulsed all over his body.

Assistance was immediately procured, and he was removed to the Leith Hospital, where the Resident on duty, Dr Goudie, recognising the nature of the injury, immediately sent off the policeman for me. I at once went to the Hospital, and was fortunate in seeing him in a convulsive seizure, which began with dilatation of the pupils and conjugate deviation of the eyeballs to the right; this was followed by spasms of the left side of the face, of the left arm, and then of the left side of the body and the left leg, followed, after an interval of at least twenty seconds, by convulsions of the whole of the right side of the body, including the face. As the seizure passed off, the eyeballs were deviated to the left side, and the pupils became moderately contracted, but no difference in their size was observed, and slight facial paralysis remained. A scalp wound 1½ inches long, leading to *un*fractured bone, was found 1½ inches above the base of the *left* mastoid process. Breathing was oppressed; pulse about 60, slightly irregular. Blood was escaping freely from the right nostril, but no blood nor fluid from either ear. Patient had three convulsive seizures while in hospital, but none so distinctly pointing to the localization of the lesion as the one I witnessed. After the fit he was unconscious, but exceedingly restless, tossing his head about.

While his head was being shaved, I sent for Dr Mill,—in the absence from town of Drs Finlay and Calder,—and on his arrival, finding that he concurred in my views as to the nature of the injury and the treatment to be adopted, I proceeded to trephine the skull at a point 1½ inches posterior to the external angular process of the frontal bone, and 1 inch above the zygoma on the *right* side. This was done under chloroform on account of his restlessness. As soon as the trephine had pierced the skull, a pro-

fuse and persistent flow of blood took place—the quantity was guessed at between four and six ounces. By the time the disc of bone was removed, the flow of blood had to a great extent stopped. The dura mater was not opened, but it was seen and heard to flap synchronously with respiration, thus showing that it was detached for a considerable area round the opening in the skull made by the trephine.

From the time of his fall until the bone was pierced, less than an hour elapsed. A circular piece of decalcified bullock's bone was inserted into the trephine opening, leaving sufficient space for drainage, and the external wound was sutured after introducing a chicken-bone drainage-tube. The patient was now quiet, with much improved pulse and respiration, had no more convulsive seizures, and woke up at 6.30 the following morning, and wished to go to work. During the changing of the dressing on that day, which was needed on account of the amount of sero-sanguinolent discharge, he was irritable, and did not remember what had occurred; but from that time he was perfectly conscious, and made a recovery only interrupted on the twelfth day by a rise of temperature (103°·6 highest point), which continued for seven days. During this time no fresh head symptoms developed, except a slight stupidity on one day; the condition of the wound was all right; nor did repeated careful examinations reveal any visceral disease, nor ophthalmoscopic examination any congestion of the discs. In this connexion I should mention that, twelve years ago, he fell into the hold of a ship, and received a severe injury on his vertex, and was unconscious and delirious for six weeks, and was unable to work for about five months.

Dr Heron Watson kindly saw him during this period of elevated temperature, and gave a favourable prognosis, which has been amply justified.

At the date of his being shown at the meeting of the Medico-Chirurgical Society (2nd December) the trephine opening was closed by a disc of firm tissue united to the edges of the opening, so that no pulsation was felt. He still has a slight degree of facial paralysis, which has persisted since the fit on the evening of the accident, but it is now barely perceptible. He states that the giddiness he suffered from since his first accident, on rising up after stooping, has since this operation almost entirely disappeared.

He resumed work in the middle of December.

Meeting III.—January 6, 1892.

Mr Joseph Bell, *President, in the Chair.*

I. Exhibition of Patient.

Dr Norman Walker showed the man with the elastic skin.

II. Original Communication.

THE SURGICAL ANATOMY OF THE BREAST AND AXILLARY LYMPHATIC GLANDS CONSIDERED WITH REFERENCE TO THE MORBID ANATOMY AND TREATMENT OF CARCINOMA: WITH A NOTE ON THE "NITRIC ACID METHOD" OF DEMONSTRATION.

(From the Surgical Laboratory of the University of Edinburgh.)

By Harold J. Stiles, M.B., F.R.C.S. Ed., Assistant to the Professor of Surgery, University of Edinburgh.

Through the kindness and courtesy of the Surgical Staffs of the Edinburgh Royal Infirmary and of Chalmers Hospital in supplying me with material, I have, during the past two years, examined over a hundred breasts excised for various diseased conditions. Almost without exception these have been subjected to a careful microscopic as well as macroscopic examination. With such a wealth of material, so generously placed at my disposal, I feel that I incur considerable responsibility, in that it is my duty to give some account of the observations I have had such a favourable opportunity of making. This I have endeavoured to do, firstly, in the Surgical Prize Essay, Royal College of Surgeons of Edinburgh 1891, and subsequently at a special meeting of the Edinburgh Medico-Chirurgical Society in January of this year, when I gave a lime-light demonstration of a series of Photomicrographs (made by Mr Andrew Pringle of London) from preparations I had made to illustrate some further observations regarding the pathology and treatment of carcinoma of the breast. Lantern slides were also shown of photographs of drawings of naked-eye specimens (prepared by what will presently be described as *the nitric acid method*) illustrating the surgical anatomy of the organ. I desire especially to thank Professor Chiene for so liberally providing the surgical laboratory with whatever was necessary to carry out the work, and for the interest he has taken in it.

A knowledge of the pathology and morbid anatomy of the mamma is essential to the correct diagnosis and successful treatment of its numerous diseases. As regards the breast, it is especially important to possess a proper conception of its anatomy under various conditions, in order to understand its pathology and adopt a satisfactory mode of treatment.

The descriptions of the mamma which are to be found in our works on anatomy do not appear to me to meet the requirements of the surgeon. I propose, therefore, in this paper to direct attention to those points in the anatomy of the mamma which are more especially of surgical importance. The methods of studying the breast hitherto employed, namely, by dissection, with or without previous boiling or partial maceration, are neither convenient nor satisfactory. The parenchyma, or gland tissue proper, is so intimately connected with the fibrous framework or stroma, that the two cannot be separated. Again, the breast tissue is not compacted into an encapsulated body, but is so broken up and branched at its periphery that the stroma becomes directly continuous with the connective tissue framework of the fatty superficial fascia. There is, therefore, no "capsule" in the ordinary sense of the term, except, perhaps, during lactation, when, however, the surgeon is seldom called upon to excise the breast. When presented to him for removal, the parenchyma relatively to the stroma and fat is scanty and difficult to recognise. In order to study the anatomy of the mamma a method is required which will reveal and differentiate to the naked eye its various constituents in an undisturbed relation, so that their form, extent, and arrangement may be accurately defined. Such an agent will be found in a mineral acid, especially nitric acid. Nitric acid was first brought under my notice practically by Mr George Brook, while I was working at the development of the mamma in the Embryological Laboratory of the University of Edinburgh. He uses a 5 per cent. to 10 per cent. solution as a histological "fixative" for embryonic tissues. Although an admirable re-agent for this purpose, I found, by subsequent experience with it in the Surgical Laboratory, that it was unsuitable for adult tissues which are at all fibrous, as it rendered them too tough for section cutting; and even if one should succeed in obtaining sections, the microscope shows that the fibrous tissue becomes swollen and hyaline under its influence. I have used the nitric acid method for the past two years in the surgical laboratory of the University of Edinburgh, and have found it a valuable aid to the study, not only of the normal and morbid anatomy of the mamma, but also of other tissues and organs, more especially when the seat of carcinoma, or of other new formations. I propose, therefore, to refer shortly to this method before passing to the surgical anatomy of the mamma.

The mode of employing it will vary slightly according to the object in view, and to the way in which it is desired subsequently to deal with the breast. The principle is as follows:—

1. Wash the mamma in water until all the blood is removed from its surface,—an important preliminary step, since the nitric acid coagulates, and blackens the blood, and thereby obscures the appearances which the method brings out.

2. Submerge in a 5 per cent. aqueous solution of acidum nitricum, B.P.

3. Wash in running water to remove the acid.
4. Place in undiluted methylated spirit.

The *rationalé* of this treatment is that the nitric acid renders all the tissues, except the fat, opaque white, due to coagulation of their albuminous constituents. By subsequently washing in water, the connective tissue becomes translucent, homogeneous, and somewhat gelatinous. Its consistence in bulk is firm, tough, and india-rubber like. The parenchyma, on the other hand, remains more or less dull grayish-white and opaque, due to coagulation of the more highly albuminous epithelial cells. The fat is unaltered. Cancerous tissue behaves in the same way as the parenchyma, and is rendered even denser and more opaque; in very cellular cancers the tissue resembles boiled white of egg, though of a grayish colour. The characteristic arrangement of the parenchyma is generally sufficient to distinguish it from the cancerous tissue.

For anatomical and naked-eye purposes, it is best to place the breast entire, or in thick slices, in the acid solution for twenty-four hours to two or three days, renewing the solution night and morning. By this means the acid penetrates the whole gland, which is then to be washed in running water for twelve to twenty-four hours. It should then be transferred to methylated spirit, which becomes brown, and must be renewed three or four times before it will remain uncoloured. In consequence of the firm and india-rubber like consistence of the stroma, clean and smoothly cut slices may readily be made through the organ; for this reason it is often better not to slice the breast until after it has been treated with the acid. The great advantage which specimens so prepared possess over ordinary spirit preparations is that the structural differentiation which is produced remains permanent. Slices of special interest may be preserved for museum purposes in spirit, and partially embedded in plaster of Paris in flat earthenware jars, covered with a glass circle. In cases of carcinoma, the relation of the tumour to the breast, its exact limits, and the mode and extent of its infiltrations, are clearly demonstrated. Excellent black and white drawings may be made of the appearances, and afford the only illustrations I have seen in which accurate detail and differentiation of structure is shown. It is now a year ago since I first introduced the method into Professor Chiene's operating theatre, and showed how it might with advantage be applied by the surgeon at the time of operation. After using the method for a few months, Professor Chiene desired me to lay it before the American Surgical Association held at Washington in September 1891, where he brought it under the notice of Professor Dennis of New York, who opened the discussion on "Recurrence of Cancer of the Breast." Professor Dennis was good enough to make the communication for me. Since then the method has been given a thorough trial both in America and in Edinburgh, and has proved of real practical value. It is employed at the operation in the

following manner:—The breast immediately after its removal is placed (after washing off all the blood) in from one to two pints of the five per cent. nitric acid solution for about ten minutes, and then washed in running water for three or four minutes. By this means the characteristic reaction is produced upon the surface, so that outlying portions of breast tissue, portions of the carcinoma itself, or small locally disseminated cancerous foci can, if here present, readily be detected. The examination can easily be completed before the time arrives for suturing the wound. The surgeon is thus afforded an additional means of ascertaining the limits of the disease and of the organ, and therefore of estimating more satisfactorily than hitherto the extent to which the parts should be removed in order to reach "an atmosphere of healthy tissue."

In by far the greater number of breasts I have examined, the nitric acid method has revealed the existence of lobules of the parenchyma on the surgeon's cut-surface, showing that the whole organ had not been removed. The reason for this will be found to be the result of an imperfect view of the anatomy of the breast. Pathologists are not agreed as to the part played by the parenchyma (when not entirely removed) in the production of "recurrence" after operations for carcinoma. The question is as important practically as it is interesting pathologically, and will receive consideration further on in the paper. It must be remembered, however, that although the surgeon's cut-surface may be free from disease, local recurrence may and often does occur as a result of microscopic or small macroscopic cancerous foci beyond the parts removed. In two cases operated on by Professor Chiene I was able to point out to him a speck of cancer, the size of a pin's head, exposed and cut across on the surface of the mamma far removed from the primary tumour. In both cases, by excising more tissue at a corresponding part of the wound the remainder of the diseased focus was removed, as was proved by microscopic examination. Not only in cancers of the breast, but in epitheliomata and cancers elsewhere, I have on many occasions found small outlying diseased areas exposed upon the cut surface of the parts removed. The application of the nitric acid test to parts removed for cancer points strongly to the fact that operative treatment in order to be successful must be more radical than is now the custom.

Having examined the surface of the breast, it should next be sliced, and again treated with the acid, in order that the naked-eye appearances of the gland may be studied. Future microscopic investigation of the parts is greatly facilitated by the above treatment, since, with the naked-eye structure clearly revealed, those portions of tissue can be selected which are most likely to demonstrate special histological points, and in this way much time and unnecessary labour is saved. For the reasons already mentioned, care must be taken that slices from which it is intended that pieces should be taken for microscopic examination are not

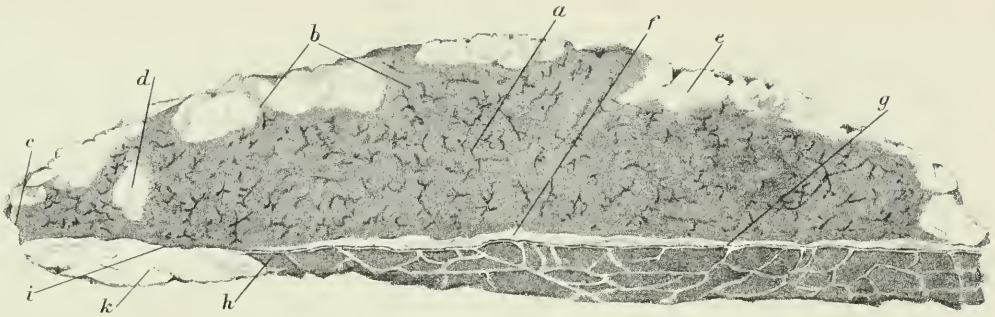


Fig. 1.

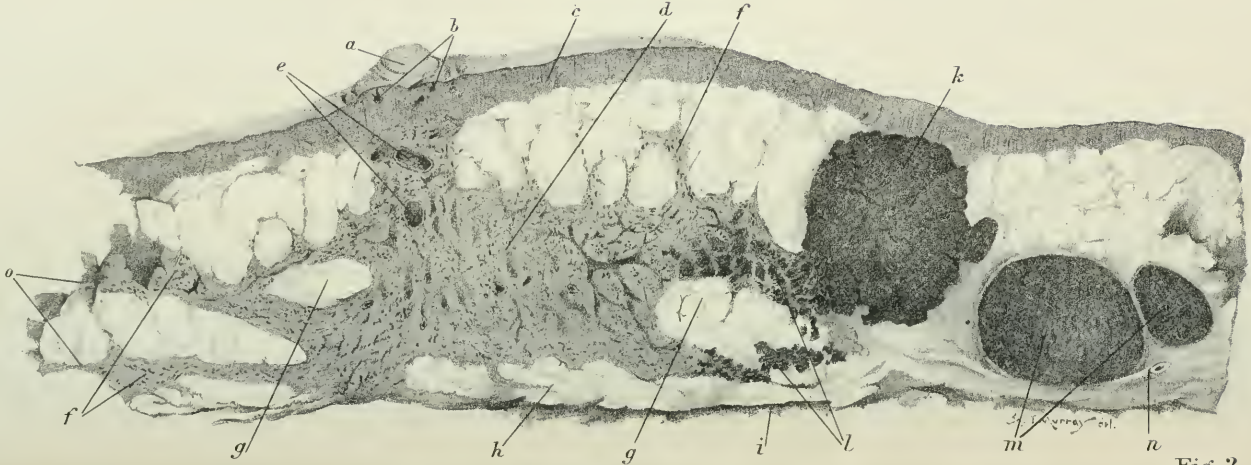


Fig. 2.

DESCRIPTION OF THE ILLUSTRATIONS.

All are drawn, natural size, from specimens treated with Nitric Acid.

Plate I. Fig. 1.

Vertical slice a little below the nipple; girl, æt. 18. (Post-mortem specimen. Skin not removed.)

a. "Corpus mammæ"—the stroma is compact, and the parenchyma is represented merely by branched ducts.
b. Peripheral processes of "corpus mammæ" ending in "ligaments" of Cooper.
c. Axillary margin of "corpus mammæ," cut across in removing the organ.
d. Intra-mammary fat lobule.
e. Subcutaneous fat.
f. Retro-mammary fat.
g. Pectoral fascia.
h. Axillary border of pectoralis major.
i. Axillary fascia.
k. Axillary fat.

Plate I. Fig. 2.

Slice from the cancerous mamma of a woman, æt. 38; married; nine children. The tumour occupied the periphery of the lower and outer quadrant; close to it was a mass of cancerous glands.

a. Nipple.
b. Sebaceous glands of areola.
c. Skin.
d. "Corpus mammæ;" parenchyma consisting chiefly of ducts.
e. Dilated ducts.
f. Peripheral processes of the "corpus mammæ."
g. Intra-mammary fat lobules.
h. Retro-mammary fat.
i. Pectoral fascia.
k. The tumour—a typical nodular scirrhus, to which the superjacent skin is tacked down.
l. Cancerous infiltration of the adjacent breast tissue.
m. Cancerous axillary glands.
n. Bloodvessel.
o. Surgeon's cut-surface (sternal edge), showing peripheral processes of "corpus mammæ" cut across.

Plate II. Fig. 1.

Slice from the cancerous mamma of a spinster, æt. 27. The tumour had been noticed 18 months, and occupied the periphery of the upper and outer quadrant.

a. Nipple and ducts.
b. Skin.
c. Subcutaneous fat.
d. Corpus mammæ.
e. Parenchyma—consisting of branched ducts from which only a few small ultimate lobules have been developed.
f. Peripheral process of corpus mammæ, ending in a "ligament" of Cooper.
g. Sternal edge of corpus mammæ cut across on surgeon's cut-surface.
h. Intra-mammary fat.
i. Pectoral fascia.
k. The tumour, which has partly invaded the superjacent skin.
l. A portion of the serratus magnus muscle to which the tumour was adherent.

Plate. II. Fig. 2.

Peripheral slice from the cancerous mamma of a spinster, æt. 39. To illustrate some points in the normal anatomy of a well-developed gland.

a. Stroma.
b. Ultimate lobules of the parenchyma arranged in grape-like clusters around the terminal branches of the ducts.
c. Connective tissue framework of circum-mammary fat (*d*).
e. Surgeon's cut-surface with (*f*) breast tissue extending up to it.

Plate II. Fig. 3.

Opposite surface of the slice illustrated in the previous figure, showing the appearance of the surgeon's cut-surface when the gland has not been completely removed. Same lettering as above.

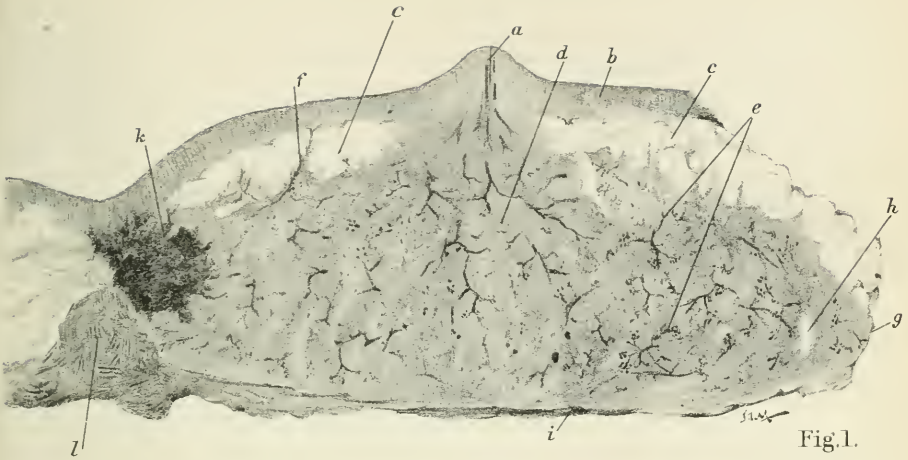


Fig. 1.

Fig. 2.

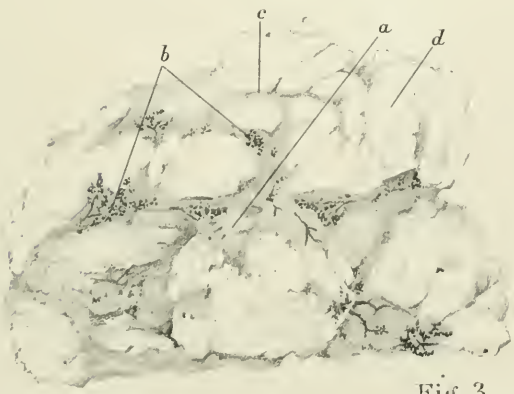


Fig. 3.

kept in the acid longer than is necessary to produce merely a surface reaction.

I now pass to consider the anatomy of the mamma from a surgical point of view.

The breast tissue consists of the following elements:—

1. The parenchyma or gland tissue proper.

2. The stroma, or connective tissue framework, which supports the parenchyma, and in which the bloodvessels, lymphatics, and nerves ramify.

3. Fat in and around the stroma.

The relative proportions of these elements vary greatly, especially according to the age of the individual, and according as the gland is, or has been functionally active. Collectively the various constituents form an organ consisting of—

(*a.*) A central part or body (the "*corpus mammæ*" of Henle[1]); (*b.*) peripheral processes.

The mamma of an adult nullipara may be taken as the type of a normal breast (Plate II., figs. 1, 2, 3). In it the corpus mammæ is well defined, and upon this the size and external configuration of the breast mainly depend. In shape it resembles very closely the ordinary bun of the pastry-cook. Its anterior surface is convex, but irregular, due to the processes which are given off from it. The posterior surface is slightly concave and more regular. The circumference is thick and well defined. If the organ be bisected vertically through the nipple in any plane, the section of the corpus mammæ presents a more or less triangular appearance, with the apex at the nipple, and the base separated from the subjacent muscle by a thin stratum of loose and delicate connective tissue. In the fresh state the stroma is tough and compact, but flabby, and of a homogeneous white appearance. The tissue of the nulliparous gland is characterised by its compact arrangement, and this is due to the small amount of fat which it contains and the close apposition of its coarse wavy fibres. Here and there an ordinary circumscribed adipose lobule, similar to that forming the subcutaneous fat, may be detected, but the more extensive admixture of fat throughout the stroma, such as is met with in multiparous breasts, does not occur. Under the nipple and areola the stroma contains no fat whatever; its fibres are loosely arranged, and allow of free mobility of the nipple, as well as of distension of the lactiferous sinuses during lactation. On close inspection, the lobules of the parenchyma may with difficulty be detected in the form of more translucent, grayish sago-like grains. In spirit preparations, the breast tissue is quite homogeneous, so that the parenchyma cannot be distinguished from the stroma. In nitric acid preparations the opaque, grayish-white parenchyma stands out in bold relief in the translucent stroma, so that its amount and arrangement can readily be studied. The ducts appear as white lines. They are

[1] *Anatomie des Menschen*, Bd. ii. p. 549.

collected into a bundle running in the vertical axis of the nipple to the summit, where they open by minute separate orifices at the bottom of a small depression. In the loose tissue under the areola they are widened out, to form the lactiferous sinuses and receive small ducts derived from the adjacent gland lobules. As the ducts extend towards the periphery of the gland they become finer and more branched, until finally, on entering the ultimate lobules they are microscopic in size. Some of the branches are prolonged beyond the body of the gland into the peripheral processes. In relation to the ducts, and scattered throughout the substance of the corpus mammæ, are numerous opaque, grayish-white, granular looking bodies from 1–2 mm. in size (Plate II., figs. 2 and 3 *b*). These are the ultimate lobules of the parenchyma, made up of microscopic flask-shaped, or short tubular gland acini opening into a common infundibulum, which leads into a terminal and microscopic duct. These ultimate lobules are aggregated more or less closely into irregular or grape-like clusters of various sizes to form larger or compound lobules, and these again are collected into lobes by the branches of a main lactiferous duct. In this way a compound racemose arrangement is produced—the lobules being much more abundant at the periphery than at the centre of the corpus mammæ. The amount of parenchyma relatively to the stroma varies considerably in different nulliparæ (compare figs. 1 and 2, Plate II.), and even in the same breast the ducts of one hemisphere or of one quadrant may possess numerous lobules, whilst the rest of the organ remains in the condition in which it is found at puberty, viz., with the parenchyma represented almost entirely by a much branched system of ducts (Plate I., fig. 1).

The peripheral processes of the corpus mammæ are relatively small in the nulliparous breast. They radiate from all parts of its surface into the circum-mammary fat. Those springing from the anterior surface appear in vertical sections of the breast as triangular tooth-like processes, with fibrous prolongations ("ligaments" of Cooper) passing from their apices to the corium (Plate I., fig. 1 *b*; Plate II., fig. 1 *f*). The parenchyma is prolonged into these processes, and in thin women reaches almost up to the corium. It follows, therefore, that the surgeon who intends to excise the whole of such a gland must either sacrifice a large amount of skin, or keep so close to it in dissecting it off the mamma as to run some risk of sloughing. From the sides of the larger processes smaller secondary processes arise, and by the junction of these with others from neighbouring processes a reticular arrangement is produced, the meshes of which are occupied by the subcutaneous fat lobules (Plate II., fig. 3). After tearing off the loose retro-mammary tissue the smooth appearance presented by the posterior surface of the corpus mammæ might lead one to conclude that it gave off no processes into the retro-

mammary tissue. As a matter of fact, however, the microscope reveals the presence of outlying gland lobules, extending from the corpus mammæ into the retro-mammary tissue, up to and between the layers of the pectoral fascia. In order, therefore, to remove all the parenchyma, the retro-mammary tissue and pectoral fascia as well as the breast must be carefully dissected off the muscle. Beyond the limits of the parenchyma the stroma of the peripheral processes becomes directly continuous with the connective tissue framework of the circum-mammary fat.

The reticular arrangement of the peripheral part of the gland is not difficult to understand, when we consider that the organ is developed by a process of continuous centrifugal budding into the subcutaneous tissue from embryonic epithelial invasions derived from the epidermis. As early as the seventh or eighth week of fœtal life, a superficial mammary area is differentiated through the invasion of the mesoblastic tissue by a solid hemispherical clump of epithelial cells produced by proliferation of the cutaneous epiblast. At the twelfth week this primary epithelial invasion begins to send downwards secondary epithelial columns equal in number to the main lactiferous ducts and lobes of the future adult gland. By the sixth month these columns have penetrated almost up to the rudimentary pectoral muscle, and are slightly branched at their extremities. They are imbedded in a well-developed fibrous stroma, which is surrounded by the developing fat lobules of the subcutaneous tissue. The stroma is continuous below with a layer of condensed fibrous tissue covering the pectoral muscle, and which sends processes between its fasciculi. During the later months of intra-uterine life the primary epithelial invasion is flattened, expanded, and cupped upon the surface, due to the horny transformation and desquamation of its more superficial cells. The secondary epithelial columns become channelled and more branched. At birth the primary epithelial invasion forms the thin epidermic covering of a circular and slightly depressed area which represents the areola, under which plain muscular fibres have already made their appearances. The nipple does not develop until a few years after birth, and is due to a papillary upgrowth of the cutis around the ducts. The rudimentary corpus mammæ at birth is a well-defined and somewhat rounded body intervening between the cutis and the pectoral muscles. The parenchyma consists of sparingly branched tubes or ducts which have not yet furnished themselves with acini. The stroma consists of compact coarse fibrous tissue surrounded by, but not mixed with fat lobules. Connective tissue processes and lamellæ radiate from the corpus mammæ to become continuous with the fibrous capsules enclosing the subcutaneous fat. From birth up to puberty the breast remains in this primitive condition, and simply grows at the same rate as the body generally. At puberty, in common with the rest of the sexual apparatus, it takes on a sudden development, which

consists in a further branching and budding out of the ducts into the surrounding tissue.

The stroma of this new parenchyma is derived from the fibrous elements of the surrounding subcutaneous tissue, the fat lobules of which remain imbedded in it. At puberty, therefore, and for several years after it, the mammæ, although to external appearances well developed, contain a relatively small amount of gland tissue which in reality represents merely the excretory ducts (Plate I., fig. 1), from which the true secreting acini have yet to be developed by a still further process of budding of flask-like diverticula around the extremities of their final ramifications. The development of the peripheral part of the gland in this reticular fashion allows of the increase in size and relatively much greater development of parenchyma which takes place during pregnancy. The evolution consists in the increase both in the number and size of the gland lobules to such a degree that the peripheral processes and lamellæ are so expanded that they become approximated, and cause more or less complete absorption of the intervening fat. In this way the fully evoluted mamma comes to possess the completely parenchymatous appearance of other secreting glands which are, and always have been, functionally active. After a variable period of functional activity the changes of involution set in by which the organ returns to its resting state. The process is characterized by an extensive atrophy of the parenchyma, and a more or less abundant development of fat cells in the persistent stroma. The breast does not return to its original virgin condition, so that one has generally no difficulty in distinguishing a nulliparous from an involuted multiparous mamma. In the latter (Plate I., fig. 2) the corpus mammæ is looser in texture, irregular, and broken up, owing to the large amount of fat in the stroma. The retro-mammary tissue has undergone a fatty transformation, so that the breast is separated from the pectoral fascia by a layer of fat of greater or less thickness. The peripheral processes are large and more widespread, so that the differentiation into body and processes is less marked. After the menopause the gland lobules undergo more or less complete atrophy, so that sometimes only the ducts remain. Small cysts (involution cysts), generally multiple, and varying in size from a millet seed to a pea, are not unfrequently met with in atrophied breasts. They are generally the result of degenerative changes in the pre-existing lobules rather than a simple dilatation of the ducts. They are, therefore most numerous upon the deep surface of the organ where the gland lobules are most abundant. They contain a serous or mucoid fluid, either colourless, yellow, brown, or bluish-green. When three or four such cysts are closely aggregated, and the intervening and surrounding tissue condensed, the condition gives rise to clinical signs closely resembling "scirrhus," and may call for a diagnostic incision before the surgeon can determine whether or not the breast should be excised. The cystic condition, however,

generally wants the characteristic density and definition of outline of scirrhus, and the rest of the breast often feels coarse and knotty, from the existence of small cysts elsewhere.

As a result of the disappearance of the gland lobules after the menopause the corpus mammæ, in spare women, becomes reduced to a more or less flattened plate-like structure, which is closely related to the subjacent muscles. The boundary-line between the body and the peripheral processes is often difficult to define. The peripheral processes are greatly narrowed and attenuated, until finally they are reduced to mere threads of fibrous tissue; they do not, however, taper away uniformly, but possess irregular and sometimes extensive thickenings at the nodes of the very irregular network which they form. This condition is not due to irregular atrophy of the parenchyma, but simply to variations in the relative amount of stroma and fat at different parts. In women who become obese after the menopause the breast tissue undergoes such an extensive fatty transformation, that in spite of the very large size of the organ very little fibrous tissue remains, except in the vicinity of the nipple and areola; the rest of the breast consists of large lobules of a yellow, oily-looking fat, between which are small irregular patches of fibrous tissue united into a very wide-meshed network by narrow bands and lamellæ in which small ducts can here and there be seen. The gland acini have almost completely disappeared. If such a breast be the seat of an ordinary circumscribed carcinoma, the tumour on palpation appears to be ill-defined, and about twice its actual size.

The surgical anatomy of the mamma would not be complete without a reference to the retro-mammary tissue and pectoral fascia; indeed, from the surgeon's point of view these structures are no less important than the breast itself, and should be looked upon as part of it. In spare women the corpus mammæ is separated from the subjacent muscles by a thin layer of loosely arranged, delicate connective tissue rich in elastic fibres; the deeper layers are more closely packed to constitute the pectoral fascia proper, which is very thin, and gives off processes which penetrate between the fasciculi of the pectoral muscle. The presence in this retro-mammary tissue and pectoral fascia of outlying lobules of the parenchyma has already been referred to. No separation can be made with the knife through this loose areolar tissue without the risk of leaving behind lobules of parenchyma. In obese subjects the retro-mammary tissue is laden with fat, which forms a thick layer separating the corpus mammæ from the subjacent muscles. The fascia in such cases is so thin as scarcely to be recognised.

The lymphatics of the breast are important alike to the surgeon and pathologist. Within the gland itself they are too small to admit of a dissection, and as they are usually collapsed in normal histological preparations, they cannot be satisfactorily studied,

unless artificially or naturally injected. Langhans,[1] although failing with senile glands, succeeded in injecting the lymphatics of non-atrophied breasts to the extent of several square centimetres by means of chance puncture injections of coloured materials into the interlobular connective tissue of the posterior aspect of the gland. The injection filled a system of lymph channels, forming a round-meshed network occupying the stroma, and enclosing one or more ultimate gland lobules in each mesh. This network receives the lymph from the interacinous spaces, and is continuous with other injected lymphatics occupying the adventitious walls of the ducts, and running parallel with them towards the areola, where they open into a horizontal plexus of larger lymphatics, lying in the loose connective tissue surrounding the lactiferous sinuses. This sub-areolar plexus (Sappey[2]) communicates freely with the lymphatics of the nipple and surrounding skin.

In carcinomatous breasts the lymphatics may frequently be seen injected, as it were, with cancer cells, so that by a careful study—more especially of the spread of the cancer in the different tissues in and around the breast—one can gain a more satisfactory idea of their structure and arrangement than is to be afforded by chance and unsatisfactory artificial puncture injections. It must not be supposed, however, that in carcinoma of the mamma the lymphatics throughout the gland are extensively and continuously filled with cancer cells. The examination of many microscopic sections, both of the tumour and of the surrounding breast tissue, may fail to demonstrate cancerous emboli in the larger lymphatic vessels. In successful microscopic preparations there is no difficulty in distinguishing the cancer cells from the epithelium of the gland parenchyma, so that the relation of the one to the other can easily be made out. The cancer cells invade first the lymph spaces of the tissue, and, since they probably possess no independent or amœboid movement, their entrance into the lymphatic vessels is more or less accidental. I have repeatedly seen lymphatic vessels containing cancer cells in all the situations in which Langhans has artificially injected them. When the perilobular and periductal lymphatics are filled with cancer cells, an appearance is produced which has erroneously been ascribed to a direct cancerous transformation or degeneration of the epithelium of some of the acini of a gland lobule. In cancerous breasts in which the tumour is closely related to the areola and nipple, the lymphatics surrounding the main lactiferous ducts are often seen to form a ring of large spaces more or less filled and distended with cancer cells. In many cases, owing to shrinkage of the cancerous emboli, the endothelial lining of the lymphatic vessels may be distinguished as a single and continuous layer of flattened cells. Outside the endothelium, the wall of the lymphatic is

[1] *Archiv für Gynæk.*, Bd. viii. p. 181.
[2] *Traité d'Anatomie Descriptive*, t. ii. p. 827, and t. iv. p. 753.

formed merely by the connective tissue surrounding the lactiferous duct. The duct itself may be more or less compressed and altered in outline, but the epithelium lining it is generally quite normal. An atrophic cancer situated close under the nipple is liable to give rise to that form of secondary infiltration of the skin which Velpeau termed "*cancer en cuirasse.*" The association of the two conditions is accounted for by the readiness with which the cancer cells may gain access to the cutaneous lymphatics of the nipple and surrounding skin through their continuity with the sub-areolar plexus.

Besides the lymphatics which are closely related to the parenchyma, there are other intra-mammary lymphatics, which, while anastomosing with the perilobular and periductal lymphatics, are more closely associated in their distribution with the bloodvessels, which, as regards the mamma, do not run in company with the ducts. If the tissue be carefully fixed, hardened, and sectioned by the paraffin process, these lymphatics when not empty and collapsed may be identified, and their structure as well as contents studied. Their structure is exceedingly simple, consisting merely of a continuous layer of endothelium, planted directly upon the unmodified connective tissue stroma which surrounds and supports the bloodvessels they accompany. I have never seen, within the mamma itself, lymphatics which possessed definite walls containing muscular fibres. The larger bloodvessels are accompanied by two or sometimes more lymphatics occupying their sheaths. When accompanying small vessels and arterioles they are usually single, of a much greater calibre than the artery, and in transverse section are often seen to surround it almost completely. In other instances the arteriole occupies the centre of a large perivascular lymph sheath. No doubt in the majority of cases the lymphatics in histological preparations are empty and collapsed; such as are not so, however, contain a delicate fibrinous coagulum, either alone or with a few leucocytes. When cancerous, they are generally filled and distended with cancer cells, which may or may not have invaded their walls and involved the surrounding tissue. Occasionally one may observe a lymphatic of large calibre, which is patent, containing only a few cancer cells which appear to have been floating along with the lymph stream. In such a condition the endothelial lining of the lymphatic is very distinct and quite normal. I have never seen any appearances which indicate that the endothelium of cancerous lymphatics in any way participates in the cancerous process.

The examination of a large number of carcinomatous breasts has afforded a demonstration of lymphatics containing cancerous emboli in one or more of the following situations, viz., in the connective tissue processes radiating from the tumour into the surrounding breast tissue or circum-mammary fat; in the breast

tissue, remote from as well as close to the tumour; in the connective tissue septa, separating the lobules of the circum-mammary fat; in the so-called "ligaments of Cooper," where they often lead to small disseminated cancerous nodules in the corium; in the retro-mammary tissue and pectoral fascia. In the last-named situation they are large, and generally accompany the bloodvessels which pass to and from the deep surface of the mamma.

With the above facts before us regarding the structure of the lymphatics, and the different situations in which we may observe them, we have now to consider the mammary lymphatic system and its circulation as a whole. Unfortunately here, as well as elsewhere in the body, our knowledge of the lymphatic circulation is very imperfect. What I have to say regarding this subject in the mamma is based upon the above anatomical facts, combined with a clinical and pathological study of the lymphatic dissemination of cancer as it occurs in this organ. Sappey describes the lymphatics of the mamma as consisting of—(1), a superficial or cutaneous set; (2), a deep or glandular set, embracing the lobules and lobes of the gland. He considers that all the trunks springing from the latter system pass from the posterior surface, and from the thickness of the gland towards the areola, where they form a plexus remarkable for the large size of its component vessels. From this sub-areolar plexus pass two, sometimes three, large trunks which empty themselves into the glands of the axilla. Langhans, on the other hand, holds that the efferent lymphatics of the true gland occupy the loose retro-mammary tissue, the larger trunks frequently following the larger bloodvessels in pairs. He has injected them simultaneously with the intra-mammary lymphatics.

My own observations have led me to look upon the mammary lymphatic system as consisting of five sets of vessels, which communicate freely with one another,—(1), A superficial or cutaneous set, including those of the nipple, areola, and surrounding skin; (2), the sub-areolar plexus of Sappey; (3), intra-mammary lymphatics; (4), lymphatics of the circum-mammary fat; (5), retro-mammary lymphatics. The cutaneous and intra-mammary lymphatics (in part) open into the sub-areolar plexus which connects these two systems. The lymphatics of the circum-mammary fat constitute a part of the general superficial lymphatic system of the chest. They receive, on the one hand, the lymphatics of the true skin, and on the other hand, efferent lymphatics from the anterior surface and circumference of the mamma. These lymphatics of the circum-mammary fat open into larger and deeper lymphatics placed between the layers of the deep fascia. Lastly, from the lymphatics of the deep fascia large trunks pierce its deep surface, and constitute the well-defined vessels with thin but muscular walls which pass to the lymphatic glands as their afferent vessels. The retro-mammary lymphatics (including those

of the pectoral fascia) receive all the efferent mammary lymphatics which leave the posterior surface of the gland. In this way, therefore, the efferent lymphatics of the corpus mammæ, of the fat around it, and of the nipple, areola, and skin over it, open either directly or indirectly into the lymphatics of the deep fascia, which latter accompany the bloodvessels of the gland, pierce the deep fascia along with them, and so reach the lymphatic glands which lie in groups or chains alongside them. The lymphatics from the inner part of the mamma accompany the perforating branches of the internal mammary artery to join the sternal glands placed along its trunk. The greater number, however, accompany the mammary branches of the acromio-thoracic, long thoracic, and external mammary branches of the axillary artery to open into the axillary glands. I cannot agree with Sappey in considering that the two or three lymphatic trunks leading from the sub-areolar plexus are the ultimate and only channels for the receipt of the lymph from all parts of the mamma. Such teaching is entirely out of harmony with what is observed both clinically and pathologically in carcinoma of the mamma.

Recurrence of the disease after operation is due to the non-removal of small and often microscopic foci of cancer, more or less remote from the main tumour, and depending for their origin upon the arrest and growth of cancerous emboli disseminating more or less directly from the primary tumour along the lymphatics. The importance of removing all the retro-mammary tissue, pectoral and axillary fascia, axillary fat and glands, along with the breast in all cases of carcinoma, cannot be too strongly insisted upon or too oft repeated. The anastomosis and intersection of the lymphatics are so free that it is impossible to say towards which set of glands the lymph from any given point in the breast will be conveyed. I have seen cancerous lymphatic emboli at the axillary border of the mamma when the tumour was situated in the inner hemisphere, and *vice versâ*. There is no doubt also that the lymphatics of the two breasts communicate to a certain extent through a median anastomosis of both the superficial and retro-mammary lymphatics. When both breasts become cancerous, one subsequently to the other, the disease in that affected later is, in the majority of cases, probably the result of lymphatic infection, and not a primary condition. In support of this view, I may instance the case of a patient under the care of Prof. Chiene, who presented herself with a second recurrence in the left breast, in the shape of a cancerous ulcer the size of a crown piece, occupying what corresponded to the lower and inner quadrant of the mammary area. There was a large cancerous mass in the corresponding axilla, and, in addition, two cancerous masses in the opposite or right axilla, one the size of a pigeon's egg, the other of a hazel nut. Further operative treatment was not deemed advisable; but, judging merely from clinical

examination, the right breast appeared to be free from cancer. In this case it would appear that the disease in the right axilla had been conveyed thither along the retro-mammary lymphatics, the breast itself having escaped.

When the breast is treated with nitric acid, according to the method already described, it becomes at once evident that the limits which our text-books on anatomy and operative surgery have accorded to the gland are not sufficiently wide, and that the breast-tissue extends much further in every direction than is generally supposed. The result is that surgeons almost invariably, though unwittingly, fall short of their intention to remove the entire gland.

The breast is described as extending from the third to the sixth (by some authors the seventh) rib in the vertical direction, and from the edge of the sternum to the anterior fold of the axilla horizontally.

Such a description would appear to be drawn, either from landmarks afforded by the external configuration and surface anatomy of the organ, or without account being taking of its peripheral processes. The extent to which the mamma overlies the axilla appears to have been more appreciated by the sculptor than by the anatomist. It must be clearly understood that the apparent surface limits of the mamma do not correspond to the extent to which the parenchyma is distributed within the subcutaneous tissue of the anterior wall of the thorax: indeed, in many instances it affords no indication of this. The peripheral processes of the *corpus mammæ* extend beyond—often far beyond—the surface projection of the breast; and this holds true in nulliparous as well as in multiparous women, though much more so in the latter.

In nulliparæ, the mamma projects at right angles from the chest. It is self supporting, so that there is no sulcus at the lower segment of its base. Its size depends on the amount of circum-mammary fat, as well as on the amount of breast-tissue proper. A breast which is hemispherical in form indicates a relatively large amount of circum-mammary fat, whereas one which is conical possesses a thicker *corpus mammæ* and a relatively greater amount of breast-tissue proper.

During pregnancy and lactation the breast undergoes considerable enlargement. The stroma is more succulent, and during involution undergoes a partial adipose transformation. After maternity, therefore, the firmness and compactness of the breast are permanently diminished, so that it tends to be pendulous, and a more or less well-marked sulcus is produced where it overhangs its base. In multiparæ who are, or who have been stout, the mammæ are very pendulous. When excising such a breast, it should be borne in mind that the base of the organ undergoes but little downward displacement, and therefore the operator must not be deceived as to the real limit of the upper hemisphere.

For clinical purposes, the mamma is subdivided artificially into four quadrants, by means of vertical and horizontal diameters intersecting each other at right angles at the nipple. The quadrants are named upper-inner, lower-inner, upper-outer, lower-outer respectively. Two adjacent quadrants constitute a hemisphere. For anatomical as well as for clinical purposes, descriptions may be rendered still more precise by the addition of two oblique diameters reaching the circumference midway between the vertical and the horizontal ones. We may employ these oblique diameters either alone or in conjunction with the others. When employed alone, they subdivide the breast into quadrants, which may be spoken of simply as upper, lower, inner, and outer respectively.

I have taken advantage of all four diameters in order to arrive at a more accurate knowledge of the anatomical relations of the base of the organ to the skeleton and muscles of the chest. For opportunities of verifying and defining the observations I had made in the operating theatre, I am indebted to Sir William Turner and Dr Symington. In the dead subject, the real circumference of the mamma may be mapped out by inserting pins in the chest wall at the points of farthest extension of the gland,—in other words, at the extremities of the four diameters. The vertical diameter extends from the lower border of the second rib to the sixth costal cartilage at the angle where it begins to sweep upwards to the sternum; the horizontal, from a little within the edge of the sternum opposite the fourth rib or interspace to the fifth rib or interspace opposite the mid-axillary line. The one oblique diameter extends from the upper border of the third costal cartilage a little without the sternum downwards and outwards to the seventh rib a little in front of the mid-axillary line; the other oblique diameter passes from the third rib a little beyond the anterior axillary fold downwards and inwards to the sixth costal cartilage midway between its angle and its sternal end. The circumference of the organ may be defined by connecting together the extremities of all the diameters. When the arm is elevated, as for an operation, the nipple in a nullipara is placed opposite the fourth rib or interspace, and only about one inch within the axillary border of the pectoralis major, thus showing that the extent to which the breast overlaps the muscle is very considerable. The level of the breasts varies somewhat according to the configuration of the chest,—thus tall women generally possess a low bust, while short, broad-chested women have usually a high bust.

The above limits, though considerably wider than those given in our anatomical and surgical works, are certainly not exaggerated; and the surgeon cannot hope to remove the whole of the breast-tissue unless in conducting his operation he keeps outside of them. It is important, therefore, to ascertain the relations of the entire mammary area to the muscles of the chest-wall, since these afford

the surgeon important guides in estimating the extent of his operation. In describing these deep relations we shall again make use of the various diameters subdividing the mammary area. The inner hemisphere rests almost entirely on the pectoralis major; at its lowest part it extends beyond the lower edge of this muscle, and slightly overlies the aponeurosis of the external oblique of the abdomen. The outer hemisphere has less simple relations, and must be dealt with in segments. The upper half of its upper quadrant rests on the greater pectoral, on the edge of the lesser pectoral, and, for a slight extent, on the serratus magnus, upon which, and under cover of the pectoralis major, it extends upwards into the axilla as high as to the third rib. Spence was in the habit of referring to this prolongation as the "axillary tail" of the mamma. The circumference of this segment crosses the edge of the pectoralis major at the level of the third rib,—that is, just where the muscle leaves the chest-wall to form the anterior axillary fold. The lower half of the upper quadrant, and the upper half of the lower quadrant, rest almost entirely on the serratus, with the exception of a small area adjacent to the nipple, which overlies the pectoralis major. The lower half of the lower quadrant has relation to the digitations of the serratus and external oblique which arise from the fifth and sixth ribs, and the part near the nipple lies on the pectoralis major. It will thus be seen that about one-third of the whole mamma lies inferior and external to the axillary border of the pectoralis major. Of this portion the upper half overlies the lower part of the inner wall of the axilla, and is separated from its contents only by the axillary fascia, which is here very fatty, so that the lymphatic glands lying imbedded in it appear to be in direct contact with the breast.

I now pass to the axillary lymphatic glands. These are so intimately associated with the mamma, both anatomically and functionally, that they call for an equal share of attention. They are described as follows in Quain's *Anatomy* (tenth edition, vol. ii., part ii., p. 556):—"The axillary glands are generally twelve or more; they vary much, however, in their number, as well as in their size, in different individuals. From four to six are placed along the axillary vessels, and receive the lymphatics which ascend from the limb; four or five small *pectoral glands* lie further forwards on the serratus magnus near the long thoracic artery, at the lower border of the pectoral muscles, and receive the lymphatics from the mamma and front of the chest, while three or four subscapular glands are situated at the back of the axilla along the subscapular vessels, and are joined by the lymphatics from the back. One or two small *infra-clavicular glands* are also found immediately below the clavicle in the hollow between the pectoralis major and deltoid muscles; they receive some lymphatics from the outer side of the arm and shoulder, and are connected above with the inferior cervical glands, below with the axillary glands. The efferent vessels of the

axillary glands ascend with the subclavian vein, and form by their union in some cases a single trunk (axillary lymphatic trunk), in others two or three large vessels, which terminate on the left side in the thoracic duct, on the right side in the right lymphatic duct. Sometimes they open separately into the subclavian vein near its termination."

In the normal condition, one is rarely able to feel any glands through the coverings of the axilla. In morbid conditions of the breast, the pectoral glands are almost invariably the first to undergo enlargement. In the later stages of carcinoma all four groups may be diseased, but generally the humeral and subscapular groups escape. Occasionally, the glands above the pectoralis minor, in the apex of the axilla, and under the clavicle, are diseased, while the pectoral glands are normal. This points to the probability that some of the lymphatics from the mamma pass to join the glands at the upper part of the axilla directly, without entering the pectoral group; and, as far as my observations go, this would appear to be the case when the tumour is central, and more especially when the skin about the nipple and areola is implicated. Some of the lymphatics which are efferent for the pectoral glands, become afferent for glands higher up towards the apex of the axilla.

When the parts which have been removed from the axilla (in connexion with mammary operations) are carefully examined, one is struck with the great variation in the appearance as well as in the number of the glands. As many as twenty, thirty, or even more may not infrequently be counted when the axilla has been thoroughly cleared out. The reasons for the anatomical underestimation of the number of glands in the axilla are, that some measure not more than one or two millimetres in diameter, while others have undergone such an extensive fatty involution as to resemble closely an ordinary fat lobule. What may be regarded as the typical glands vary from about the size of a grape stone to that of a small pea. In the region of the hilus the lymphoid tissue becomes replaced by a greater or less amount of fibrous tissue, which serves to support the main divisions of the blood-vessels and efferent lymphatics; and from this connective tissue processes are given off which ensheath the vessels, and in their turn give origin to the delicate connective tissue recticulum, in which the leucocytes of the follicular parts of the gland are entangled. The reticulum occupying the lymph sinuses is produced from the retiform branching of delicate connective tissue processes which are given off from the fibrous trabeculæ penetrating the gland from its capsule. The afferent and efferent lymphatics lose their adventitious and muscular coats where they respectively open into and out of the lymph sinuses, the endothelial lining of which is continuous with that of the lymphatics. If sections of the gland which have been stained with nuclear dyes be examined under low magnifying power,

there will be observed, scattered here and there throughout the gland, rounded foci or masses of cells which have stained less deeply than the follicular tissue generally. These are the germ centres (Keimcentren) of Flemming.[1] They are always embedded in the lymphoid follicles, or in the lymphoid cords of the gland, and they are most numerous in the cortical region. When more highly magnified, the cells that form the centres are seen to be larger than the densely crowded lymphoid cells that surround them. A closer comparison of the two kinds of cells shows that those forming the germ centres possess larger and less deeply stained nuclei, and a greater amount of perinuclear substance. These are fully developed leucocytes, in many of which mitotic figures may be seen: in other words, they are centres of proliferation, the daughter cells—the small lymphoid cells—having migrated and become crowded together around them. These lymphoid cells find their way into the lymph sinuses, and ultimately reach the blood as young leucocytes.[2] In examining axillary glands for malignancy, it is important that one should be familiar with these germ centres, otherwise they might be mistaken for cancerous foci.

Having referred to the essential points in the microscopic anatomy of a gland, I wish now to describe one or two departures from the normal type. Although these have been studied mainly from material derived from patients suffering from carcinoma of the mamma, I may state that I have also met with similar conditions apart from cancer, and in other regions of the body; for example, in the neck, the anterior mediastinum, the groin, and the popliteal space. No doubt they occur throughout the body generally.

In the first place, the very minute glands, which do not exceed one or two millimetres in diameter, possess a very primitive structure, and afford a clear demonstration of the essential parts of a gland. Here we have to deal merely with a single lymphoid follicle, between which and the connective tissue capsule is an unbranched lymph sinus connected with only one afferent and one efferent lymphatic. A germ centre may sometimes be observed in the centre of the follicle, thus proving that these little glands are functionally active. They correspond closely to the endolymphangial nodules which Klein[3] has described as occurring in the serous membranes. They differ from the solitary glands of the alimentary and respiratory mucous membranes in the possession of a distinct capsule, which is doubtless the thickened wall of the original vessel, inside of which the follicle has developed.

[1] "Studien über Regeneration der Gewebe," *Arch. f. Mikr. Anat.*, vol. xxiv., 1885.

[2] For further details as to the functions and origin of these structures, Gulland's paper "On the Function of the Tonsils," *Edin. Med. Journal*, 1891, may be consulted.

[3] *The Anatomy of the Lymphatic System*, part i.

Secondly, the glands, which have undergone a more or less complete fatty involution, are found in variable numbers during the resting state of the mamma, both before and after the menopause. Their size also varies greatly. They may be no larger than a grape stone, or they may reach the size of a filbert, or may be even larger. Their number and size are greatest in women who have become obese after the menopause, and whose breasts have therefore undergone very extensive fatty transformation. The naked-eye appearance of a bisected fatty involuted gland is peculiar and characteristic. It differs markedly both from that which is presented by a typical lymphatic gland on the one hand, and by an ordinary fat lobule on the other. The hilus is represented by a more or less distinct notch, opposite which, and immediately under the capsule, is a narrow crescentic layer of lymphoid tissue, occupying the whole or a greater part of the circumference of the gland. The extremities of the crescent often curve into the substance of the gland at the hilus. This lymphoid tissue possesses a uniform grayish-pink and fleshy appearance, resembling granulation tissue. Within the lymphoid crescent, and forming the bulk of the gland, is the fatty portion, which is softer and more bulging. It differs, however, from ordinary fat, both in appearance and in consistence. It is firmer and has a pale grayish colour, almost without any yellow tint, and with but little of the oily aspect of ordinary fat. The interpretation which has, I believe, frequently been given of these appearances is that the firmer, more vascular, grayish-pink concentric zone represents the cancerous portion of a malignant gland, while the fatty medullary portion is looked upon as the remains of the normal gland tissue. Microscopic examination, however, at once reveals the error. At first sight one gets the impression that the structure is not a lymphatic gland at all, but is merely an ordinary fat lobule, the peripheral portion of which is occupied by an inflammatory leucocyte infiltration. Further investigation shows that every stage of transition exists between almost complete fatty metaplasia and what may be looked upon as a typical lymphatic gland. The earliest sign of fatty involution is evinced by a fatty metaplasia of the cells of the connective tissue in the region of the hilus; next, the connective tissue framework of the medullary part of the gland becomes involved; and, finally, the whole gland, with the exception generally of an incomplete and very thin layer immediately under the capsule, becomes converted into adipose tissue. The lymphoid tissue that remains is always the cortical portion, the follicles of which may still contain germ centres, showing that the gland is not completely functionless. A distinct lymph sinus (subcapsular sinus) containing a retiform network intervenes between the capsule and the lymphoid tissue, and is continuous with other sinuses that surround the connective tissue trabeculæ penetrating the gland from the capsule. These, again, are continuous with lymphatic channels which run from the

inner margin of the lymphoid zone through the fat towards the hilus, where they unite to form wider channels which emerge at the hilus as the efferent vessels. Afferent lymphatics may be seen opening into the subcapsular sinus, just as in an ordinary gland. The microscopic appearances of the fat differ from that of an ordinary fat lobule, not only in possessing many large lymph channels, but also in the correspondingly large number and size of the bloodvessels which pass to and from the hilus. The presence of so many large blood and lymph vessels in the fat accounts for the more porous, more vascular, more serous, and less oily appearance which distinguishes it from ordinary fat.

In order to understand the manner in which this fatty involution or metaplasia is brought about, and the conditions in which it occurs, we must remember that these lymphatic glands are associated both functionally and anatomically with the mamma. In a former part of this paper I showed that the mamma undergoes important structural alterations in relation to its various functional states. The process of involution of the mamma, whereby it passes from the condition of full functional activity to a state of rest, is attended not only by partial atrophy of the parenchyma, but also by a conversion of much of the connective tissue into fat. After the menopause, the more or less complete atrophy of the parenchyma is, except in spare women, accompanied by a still further fatty transformation of the stroma; and in obese women very little of the original stroma remains.

The fatty involution of the axillary glands corresponds exactly to the process above described in the mamma; that is to say, we have an atrophy and absorption of the follicular tissue, along with a fatty metaplasia of the connective tissue framework.

During the functional state of the mamma, the lymph which is drained from it to the glands is greater in quantity and altered in quality as compared with its resting and atrophied state. There is an increase both in the deposit and in the production of leucocytes in the lymph glands, the increased production being shown by the large number and size of the germ centres present.

During the resting condition of the mamma, and still more after the menopause, the deposit and production of leucocytes within the lymphatic glands are very slight. More leucocytes are washed out of the glands by the circulation of the lymph through them than are conveyed to them or manufactured in them. Accompanying this decline of function, and no doubt in some way associated with it, is the fatty transformation of the connective tissue framework. The cortical part of the gland, which is always the most active functionally, is the last to undergo the change.

This fatty involution or metaplasia of lymphatic glands appears to me to afford a field for speculation and further study, especially in its possible relations to such diseases as lymphadenoma, tubercle, syphilis, etc. It is not difficult to account for the large number

of glands which come into prominence in these diseases, as compared with the number which anatomists describe as existing normally, if we consider that many of the glands which have become enlarged are such as had normally undergone fatty involution, but which, as a result of the irritation to which they became subject in these diseases, have again become evoluted and diseased.

The fatty involuted axillary glands are of interest to the surgeon both clinically and pathologically. As a rule, palpation through the coverings of the axilla gives no indication of their presence, although occasionally, when large and numerous, they give rise to what is often spoken of as a "fulness in the axilla." They are usually discovered when the axilla is explored from the wound made to excise the breast, and are then spoken of as "enlarged glands," and, as I have already pointed out, are either regarded as malignant, or are looked upon with the greatest suspicion. In stout women, their resemblance to an ordinary fat lobule is such that they are liable to be overlooked by the surgeon who is content with exploring the axilla with the view of discovering and "shelling out" only those glands that are distinctly indurated. During the operation, they may be distinguished from fat lobules by three positive characters, viz,—(1.) When grasped between the finger and thumb they are firmer and more elastic; (2.) They possess a distinct capsule which enables them to be readily shelled out from the surrounding fat; (3.) During their removal, the blood and lymph vessels connected with them are brought into view, and have to be torn or cut across before they can be completely separated. The larger they are, the thinner and more stretched is the capsule. Their greater firmness and elasticity as compared with an ordinary fat lobule are due to the greater tension under which the fat and the lymph are confined within the capsule.

When one of these glands is treated with a five per cent. solution of nitric acid, the cortical zone of lymphoid tissue becomes dense and opaque white, and remains so after being subsequently washed in water. The fatty portion is unaltered. By this means, therefore, the true nature of the gland is clearly revealed. In a previous part of this paper I showed the value of the nitric acid method in differentiating cancerous tissue in the breast. It is necessary to point out, however, that the same method does not render the detection of cancer in a lymphatic gland any easier than it would be in the fresh and natural condition, since the lymphoid tissue, on account of its highly cellular and therefore also highly albuminous nature, reacts to the nitric acid in much the same way as cancerous tissue.

Microscopic examination of fatty lymphatic glands, which have been removed along with a cancerous mamma, shows that the fatty part of the gland is being invaded by leucocytes, especially along the lines of the blood and the lymph vessels. This leucocyte infiltration is accompanied by a connective tissue and endothelial

proliferation, especially noticeable at the invading edge. It would appear, therefore, that the irritation and cell activity which is going on in the mamma so affects the axillary glands as to produce an evolution in them, whereby they become converted again, more or less completely, into lymphoid tissue. The transformation, when complete, results in what is spoken of as a "simple" enlargement of the gland. The simple enlargement of the glands, which is often associated with the presence of simple neoplasms, cystic disease, chronic mastitis, etc., is no doubt brought about in the same way from glands which have undergone more or less fatty involution. The same glandular enlargement may be observed in other regions of the body, when the parts from which the lymph is derived are under similar pathological conditions.

Some surgeons are inclined to look upon all glandular enlargements associated with carcinoma as necessarily malignant. Such a view, while incorrect pathologically, errs on the safe side from the therapeutical point of view. Professor Chiene has long been in the habit of pointing out that this simple enlargement of lymphatic glands is especially liable to be associated with malignant disease when attended with ulceration and sepsis, and that when the tumour is removed the glandular enlargement disappears. I have on several occasions microscopically examined enlarged (often much enlarged) glands removed from the axilla, neck, and groin, in cases of malignant disease of the breast, tongue, and penis respectively, without discovering any cancer in them. Such enlarged glands may or may not be the seat of acute or chronic inflammation. When acutely inflamed they are only slightly indurated, of a red or reddish-purple colour, and often mottled with minute hæmorrhages. Vascular engorgement, rupture of capillaries, and choking of the lymph sinuses with leucocytes and red blood corpuscles, express the microscopic appearances. Simple enlargement with chronic inflammation gives rise to greater induration; the inflammatory hyperplasia affects especially the connective tissue framework of the gland, which is often seen to have undergone a marked hyaline degeneration. The lymph sinuses contain proliferated connective tissue and endothelial cells, rather than leucocytes. Glands of this description are frequently found where the breast is the seat of a simple tumour of cystic disease and of chronic mastitis. In the circumscribed variety of the latter disease their presence only serves to add in some cases to the difficulty of distinguishing it from carcinoma.

While I have hitherto spoken of the fatty glands as originating only from pre-existing glands, I am not prepared to say that this is the only way in which they are produced. On the contrary, there is evidence to show that they may possibly also be developed simply from fat lobules, which are more closely related to the lymphatics than usual, in the same way that some fat lobules possess a distinct afferent artery and efferent vein

united by a capillary network occupying the substance of the lobule. In the breast itself, especially when the seat of a rapidly infiltrating carcinoma, I have certainly observed the formation of lymphoid nodules and germ centres in connexion with the perivascular lymphatics of a small artery and vein, which occupied a delicate connective tissue lamella of the intra-mammary fat. A similar new formation of lymphoid tissue occurs sometimes at certain points around the vessels and lymphatics which lie between the fat lobules of the axilla.

Lymphatic glands, when cancerous, are frequently so extensively diseased that no trace of the original lymphoid tissue remains. When this is the case, they are characterized clinically by marked induration, and usually by enlargement. Occasionally several cancerous glands are matted and conglomerated into a mass of the size of a child's fist. In cases where the disease is confined to the medullary part of the gland, the cortical region responds to the irritation produced by the cancer, the result being an increase in the number and size of the germ centres, and a dense accumulation of lymphoid cells (young leucocytes) around them. All the varieties of lymphatic glands I have referred to may be cancerous,—that is to say, may contain cancer cells. Theoretically the initial stage of the disease consists in the deposit in the gland of a single cancer cell, which has been conveyed to it from the cancerous mamma along the lymphatics. Although it is practically impossible actually to demonstrate this, one may nevertheless observe the condition in which only a few cancer cells exist in the gland; they generally occupy the subcapsular sinus, that is, just the place where anatomically we should expect to find them. I possess a microscopic section of a fatty lymphatic gland showing this state of affairs. The preparation was made to show the normal structure of the gland, which, to the naked eye, appeared to be absolutely healthy. Such a gland, in consequence of the fatty change which it has undergone, feels even softer than a normal gland when made up entirely of lymphoid tissue. It follows, therefore, that the absence of induration does not always signify freedom from malignancy. Again, it must be remembered that the smallest glands are sometimes malignant. As in this condition they may be no larger than an ordinary pin's head, the absence of enlargement does not necessarily imply non-malignancy.

The main lymphatic trunks of the axilla run for the most part alongside of the bloodvessels. In consequence, however, of their small size and the thinness of their walls, it is difficult to display them by dissection. The afferent vessels of a gland are more numerous, but smaller than the efferent. The microscopic structure of the wall of a lymphatic trunk closely resembles that of a small vein. Indeed, when they are isolated and empty it is sometimes difficult to say with which one is dealing. If, however, the lymphatic is lying side by side with bloodvessels,

it may be recognised by its more or less collapsed, longitudinally folded, and relatively thin wall. The lumen is small and stellate, and is either empty or contains a little granular matter and a few leucocytes. Judging from the extent to which the wall is folded, the calibre of the vessel when distended must be very large relatively to the thickness of the wall—a relationship which appears to hold true throughout the lymphatic system generally. As regards the coats of the lymphatic, one fails to detect the well-marked differentiation between the muscular and the adventitious layers. In the small arteries the middle coat is purely muscular and the outer purely fibrous. In the veins there is a considerable admixture of fibrous and elastic tissue between the muscular fibres of the middle coat. In the lymphatic trunks the whole thickness of the wall outside the internal coat is made up of a mixture of muscular and fibrous tissue in about equal proportions. The muscular fibres are collected into small bundles separated from one another by the loosely felted white and yellow connective tissue fibres. Another distinguishing feature of the lymphatic is that the muscular fibres, instead of all running circularly, as in the case of the bloodvessels, run many of them longitudinally. The majority of the circular fibres lie internal to the longitudinal, but frequently the two sets of fibres are mingled. Valves, consisting of two opposed segments, are placed at frequent intervals along the main lymphatic trunks. They consist of a reduplication of the endothelial layer of the vessel. A transverse section opposite the valve gives the appearance of a treble lumen to the vessel, the middle compartment being bounded on either side by the section of a segment of the valve which is so extensively pleated as to present a very zig-zag edge, thus showing how greatly distended the vessel would have to become before the valve became incompetent.

The important part played by the lymphatics in carcinoma of the mamma has already been referred to. Surgeons are in the habit of indicating their cognisance of this fact by sometimes referring to a "thickening" of them, which may be felt, not only when the axilla is opened up, but even through its coverings. As the result of the examination of the parts removed from over a hundred axillæ in cases of cancer, I am satisfied that the lymphatics are in no way responsible for this stringy feel. The condition is felt most distinctly in spare women with more or less atrophied breasts, and is due to the presence of the ducts of the breast tissue, which run like so many branched strings under the edge of the pectoralis major, and upon the surface of the serratus as high up in the axilla as the third rib. They are, in fact, the ducts belonging to what Spence used to speak of as the "axillary tail" of the mamma. That this is the case may readily be demonstrated by stripping off the fascia from the posterior surface of the breast (with which the

axillary tissue is left in contact), and then by applying the nitric acid method to it. The structures which give rise to the stringy feel will appear as opaque white lines, which may occasionally be traced as far as the cancerous glands. Microscopic examination shows that they are mammary ducts and not lymphatics. One may examine both macroscopically and microscopically the tissues from many cancerous axillæ without being able to discover any disease whatever in the lymphatic trunks. It is evident, therefore, that as soon as the cancer cells reach lymphatics of any size, they are swept along them to the glands, in the same way that an embolus is washed along a vein. The lymphatics do not always escape in this way, because stationary cancerous emboli are frequently present in the smaller vessels, and of course can only be discovered by microscopic examination. They subsequently invade the wall of the lymphatic, and give rise to nodules of cancer which are distinct from the lymphatic glands. In the comparatively few cases in which I have found the main trunks diseased, the cancer cells could always be traced as far as a malignant gland, the lymph sinuses of which, in consequence of being blocked, had no doubt caused a heaping up of the cancer cells behind them.

It is very doubtful whether a cancerous condition even of the largest axillary lymphatics can be detected during the operation. Examined microscopically, the wall is seen to be thickened—a condition due to chronic lymphangitis. The lumen is filled with cancer cells. To the naked eye, therefore, such a diseased lymphatic resembles a small bloodvessel which has been plugged or obliterated.

After describing the anatomy of the parts concerned in carcinoma of the mamma, a number of photographs were shown to illustrate the manner and extent of their involvement in the disease. As regards the tissues extrinsic to the mamma, viz., the skin, subcutaneous fat, pectoral fascia, and muscles, the preparations show conclusively that their diseased condition consists in the presence of cancer cells here and there in their lymph spaces and lymphatic vessels. The cancer cells do not originate from the cell elements of these tissues; they are the descendants of pre-existing cancer cells, and have been conveyed thither by the lymph stream, and occasionally also by the bloodvessels. The inflammatory connective tissue hyperplasia, attended with more or less leucocyte exudation, is a secondary element in the disease. The term "recurrent," applied to the disease which manifests itself after operation in these extrinsic structures, is obviously a misnomer, since what really occurs is a continuance of the original disease which was already present at the time of the operation, and which in the course of its life history becomes, from a structure visible only with the microscope, a palpable nodule. I have already shown how seldom all the gland tissue is removed in operations for cancer of the breast. The question, therefore, arises, "What part does

the breast tissue that has been left behind play in the production of recurrence?" Surgeons are not agreed on this point, and herein lies the difference of opinion that exists regarding the necessity of removing the entire breast in all cases of carcinoma.

Mr Butlin,[1] in arguing against radical operations as a *sine qua non*, says,—"Certainly in the majority of instances there is nothing to lead one to believe that the new growth arises in the outlying lobules of the mammary gland, or in any remains of the parenchyma of the gland." Heidenhain,[2] on the other hand, in a very admirable paper, read before the German Congress of Surgeons in Berlin in 1889, maintains that in carcinoma of the mamma there are proliferative changes in the lobules throughout the whole gland, which must be looked upon as the direct forerunner of cancer ("das mittelbare Vorstadium der Krebsentwickelung"), and that sooner or later will pass into typical cancer.

My own observations on the causes of recurrence of cancer of the breast lead me to take up an intermediate position. I hold that when cancer manifests itself in breast tissue which has been left behind, the disease originates in the majority of cases from pre-existing cancer cells derived directly or indirectly from the original tumour, and occupying the lymph spaces or lymphatics of the stroma. In other words, "recurrence" in breast-tissue which has been left behind originates in the same way as "recurrence" in the tissues extrinsic to the mamma and in the axilla. While admitting that recurrence may originate from the epithelium of mammary acini which has been left behind (just as the original tumour has arisen from mammary epithelium), I maintain that it does not necessarily do so; on the contrary, I believe it to be a quite exceptional cause of recurrence. I am familiar with the various conditions which Heidenhain[3] refers to as existing in the acini and lobules, both adjacent to the tumour and to the "surgeon's cut-surface." The conditions which he looks upon as pre-cancerous present histological appearances which are very different from those of cancer proper. In the case of many breasts they are not only altogether absent, but when they do occur the most careful examination of all parts of the organ fails to detect the transitional stages of their development into cancer; moreover, these so-called pre-cancerous conditions are also met with in breasts which are the seat of chronic mastitis, cystic disease, etc. I would therefore advocate that the principle which should underlie all operations for carcinoma of the mamma (or carcinoma wherever situated) is the complete removal, not only of the tumour and the organ in which it lies, but also of as much of the surrounding tissues as is likely to contain the lymphatic spaces and highways along which

[1] *The Operative Surgery of Malignant Disease*, p. 378.

[2] "Ueber die Ursachen der localen Krebsrecidive nach Amputatio Mammæ," *Verhandlungen der Deutschen gesellschaft für Chirurgie*, Berlin, 1889.

[3] *Loc. cit.*

the malignant elements of the disease (as far as our present knowledge goes—the cancer cells) have been disseminated. Unfortunately, it is impossible in any given case to say to what extent this may have taken place. Here exactly lies the difficulty and uncertainty of the operative treatment of malignant disease. Much, however, may be done by a more careful study of the histological relations of the tumour elements to the organ, and especially to the lymphatics of the part in which it is situated. In the present state of our knowledge the surgeon will do well to err on the side of sacrificing too much rather than too little tissue.

Professor Dennis,[1] in his paper on "Recurrence of Carcinoma of the Mamma," admirably puts the case when he says,—"The recurrence of carcinoma of the breast is influenced by the radical character of the operation itself. No procrustean rule can be laid down in regard to the extent or character of the operation for the removal of carcinoma of the breast which would meet the exigencies in every case. There is, however, a standard operation which is none too severe to meet the necessary conditions in every case. The uniform classical operation should include the entire breast gland, all the fatty areolar connective tissue in the vicinity, the integuments over the circumscribed area of the tumour, and as much more as is necessary, leaving out of consideration altogether the question of flaps to cover the wound, and, finally, the pectoral fascia." In another part of the paper he again refers to this as follows:—"I am a strong advocate of always removing in every case, to which there is no exception, the entire breast, with the pectoral fascia and the lymphatic glands, as the minimum operation in the most insignificant scirrhus. The operation in nearly all cases must extend beyond the limits of the breast, and include a most radical one."

It is worth while to inquire how this "standard" and "minimum" operation should be carried out in order to give the best chance of removing all the disease with, at the same time, the least amount of mutilation. The first and perhaps the most important step in the operation is to make suitable skin incisions. Although these must vary with the position of the tumour and the amount of skin invasion, there is nevertheless a definite principle to be followed in all cases, and that is so to plan the incisions as best to facilitate the removal of the entire organ, and at the same time of whatever skin is diseased or is at all likely to be so. Let us take, for example, the case of an ordinary circumscribed scirrhus, with the skin over it not obviously involved. According to Gross,[2] the plan to adopt would be "to carefully palpate the entire mammary region while the patient is supine, in order to discover any outlying lobules, should they exist, and then with an aniline pencil to draw a line round the entire circumference of the

[1] *Trans. of the American Surgical Association*, vol. ix. 1891, pp. 226 and 229.
[2] Mann's *System of Gynecology*, vol. ii. p. 314.

breast as a guide for the knife. If the tumour be peripheral, the incision must extend for at least one inch beyond its apparent limit. A stout large knife is then carried along the line down to the pectoral muscle." Such a method, in my opinion, is open to great objection. In the first place, from what has been said of the arrangement and extent of the breast, it is obvious that the circumference of the gland cannot be defined by palpation. Gross's "circle" will be considerably smaller than that formed by the real circumference of the gland, so that to cut directly down along it to the pectoral muscle would necessarily leave behind more or less of the peripheral processes of the gland. Such a method, besides leaving behind breast-tissue, and removing too little of the circum-mammary fat, is open to the objection that it sacrifices an unnecessary amount of skin. In a simple case, such as that we are referring to, it is quite unnecessary to remove a large circle of skin. The old elliptical incision, if sufficiently large, is the best incision to employ, so far as the removal of the entire gland is concerned, and it should be made parallel to the oblique diameter of the mamma, its lower extremity reaching well down below the costal margin close to the ensiform cartilage, and its upper extremity terminating at the outer border of the pectoralis major opposite the third rib. The ellipse should be widest opposite the nipple, and should there measure at least four inches across. In stout patients it can be made much wider, and will still allow the edges of the wound to be brought in contact. In an obese patient recently operated on by Professor Chiene, the edges came together without difficulty, after the removal of an ellipse measuring twelve by eight inches.

The axilla is laid open by carrying an incision from the outer extremity of the ellipse along, or a little below, the edge of the pectoral across the axilla to its outer wall, as far as the angle between the lower edge of the tendon of insertion of the pectoral and the inner border of the biceps. The axillary incision should be made at the same time as the ellipse, and both should extend down to, but not through, the subcutaneous fat. If the ellipse includes the whole of the skin over the tumour, the surgeon may at once proceed to reflect the flaps off the breast; but, if the tumour does not fall within the ellipse, the skin over it should be removed by a triangular incision (as recommended by Dr Joseph Bell, *Edinburgh Medical Journal*, 1871), the base of which forms part of the ellipse. This plan should be followed not only when the skin is evidently involved, but even when it is freely movable and apparently healthy. It will be obvious that unless such a method be adopted, the ligaments of Cooper, containing lymphatics in direct continuity with those about the tumour area, and therefore liable to contain cancer cells, will be left, and consequently recurrence in the skin or subcutaneous fat is liable to occur. I believe neglect of this

procedure is often responsible for the superficial recurrent nodules which are not infrequently observed in and close to the cicatrix. A combination of these incisions will be most suitable in the majority of cases in which the tumour is peripheral—the result being a T-shaped cicatrix. The lower and outer flap should first be dissected off the breast, keeping as close to the skin as is consistent with the maintenance of its vitality. This dissection should be carried as low down as to the seventh rib in the mid-axillary line. The upper and inner flap is reflected inwards beyond the edge of the sternum, and upwards almost as high as the clavicle. It is to allow of this free dissection that we recommend that the axillary incision should be made at the same time as the ellipse. The breast, with the exception of the axillary portion, should now be freed from the chest wall along with the fascia of the muscles which underlie it, viz., pectoralis major, serratus magnus, and obligus externus. Unless the upper digitations of the latter muscle be laid bare, the probability is that some peripheral processes of the gland have been left. The axilla is dealt with by first cleaning the edge of the pectoralis major as far as the biceps; in this way the axillary fascia is divided and the axilla opened into. The axillary vein is exposed at this stage lying immediately underneath the fascia at the outer extremity of the incision. The fat and glands should be removed as far as possible *en masse*, by working in the first instance from the edges of the pectorals back to the latissimus dorsi so as to expose the serratus magnus and subscapularis. The intercosto-humeral nerves, which come into view at this stage of the operation, may be avoided, and care should be taken not to injure the two lower subscapular nerves. The dissection is then continued along the inner wall under the pectoralis minor to the apex of the axilla. Finally, the fat and any glands are to be separated from the axillary vessels; and care must be taken that branches of the axillary vein which come into view are not torn through close to the parent trunk.

As regards the treatment of the wound, if the operation be conducted according to aseptic principles, no drainage tube is required, and the case will generally not require to be dressed until the end of a week, when the stitches may be removed. If, however, strong antiseptics have been employed to douche out the wound, both before and after stitching, the copious serous exudate caused thereby necessitates, in my opinion, the use of a drainage tube for the first twenty-four hours. The above operation is, I believe, sufficiently radical for simple cases.

When, however, the skin over the tumour is involved, its removal must include a wide margin of apparently healthy skin. When, in addition to being invaded in the region of the tumour, the skin is also the seat of multiple nodular cancerous disseminations, then the whole or nearly the whole skin over the mamma must be removed, since, in addition to the palpable nodules the *nitric acid*

method shows that there are other disseminations beyond them which are too small to be recognised by the most careful clinical examination. Professor Watson Cheyne[1] has shown the great value of grafting skin upon the pectoral muscle at the time of the operation when the wound cannot be closed. In one of his cases, in which the gap measured 8 by 6½ inches, the whole wound was soundly healed within a month. If the tumour, as is so frequently the case, be adherent to the pectoral fascia, a large piece of the subjacent muscle should be removed, even although it should appear to be quite healthy. The neglect of this procedure results not only in the exposure of a part of the surface of the tumour on the "surgeon's cut-surface," but also in the likelihood of a few cancer cells being left behind on or between the superficial fasciculi of the pectoral muscle.

When the pectoral muscle is obviously invaded by the tumour-mass, and by nodular disseminations in the vicinity, the prognosis will be most unfavourable. Heidenhain points out that the contractions of the muscle favour the spread of the cancer cells along the lymphatics, and recommends that the entire muscle be removed, but this appears to me to be unnecessarily radical.

Lastly, it will be gathered from what has been said regarding the pathology of recurrence, that a knife which has once cut into cancerous tissue should not be used again in the same operation.

The President, in returning the thanks of the Society to Mr Stiles for his most interesting, monumental, and exhaustive address, rendered so valuable and intelligible by the beautiful limelight illustrations, expressed his great satisfaction with it. Its wide range rendered it interesting to the embryologist, the pathologist, and the surgeon, and a wide field of discussion was opened. From his own point of view, as an operating surgeon, he agreed entirely with Mr Stiles' argument, that not only should the whole breast be removed with scrupulous exactitude in every case of cancer, but also much skin over it, the whole pectoral fascia, and as much as possible of the contents of the axilla. He absolutely dissented from an opinion he read lately in a southern authority, that the pectoral fascia should if possible be left intact, as the healing was made easier and septicæmia was less likely to follow. Before sitting down, he mentioned, for the encouragement of surgeons, that within the last week he had seen two cases of ladies, each of whom had had both breasts removed at different times for cancer, and both of them were in perfect health, one eight years after the second operation.

Professor Chiene expressed the great interest with which he had

[1] *Lancet*, 4th July 1891.

listened to Mr Stiles' demonstration. He had for two years observed day by day the investigation as it progressed. As the work was done in the laboratory, the methods devised by Mr Stiles were applied clinically; and the speaker very strongly recommended that the nitric acid method should be used at the operation, and frequently it would be found that the operation had to be extended. If this method was used, much help would be given to the surgeon to be more thorough in the operation, and give greater hope of a permanent cure. Professor Chiene expressed the great gratification with which he had heard this demonstration, founded on work done in the department over which he had the honour to preside.

Mr A. G. Miller had heard Mr Stiles' paper with much pleasure, and had learned many things from it which he intended to put in practice. He would much rather think over what he had heard than speak about it. He would like to say, however, that he had already been able to carry out some of Mr Stiles' suggestions, which he had received from him from time to time at the Royal Infirmary. That good results have followed excision of the breast not unfrequently is known to all hospital surgeons. Mr Miller recently removed the right breast from a woman whose left breast had been removed by the President eight years before. The result on the left side was perfect. Mr Miller had also removed some years ago a small nodule that had appeared at a stitch-hole in another patient of the President's. The irritation of the stitch had been the determining cause of the return in this case. The patient was heard of recently as well.

Surgeon-Major Black stated that he had seen the practice in Middlesex Hospital some years since of Dr Fell's system of enucleation of cancerous tumours of the breast, which met some measure of operative success. He was not, however, aware what success was obtained in preventing a recurrence of the disease; but it was impressed upon the profession that the powder or paste would only have its full caustic effect upon the cancerous tissues. The first reports on this method of treatment will be found in the Reports of the Middlesex Hospital about 1857–60.

Mr Cathcart asked Dr Stiles if he had observed any relation between the position of the primary cancerous nodule and the secondary deposits in the gland itself? If these were due to intramammary lymphatic extension, they ought to be more numerous when the primary deposit was at the source of the mammary lymph stream than when it lay near where the lymphatics left the mamma. Thus, if the axillary lymphatics left at the axillary border of the mamma, a primary nodule there ought not to cause secondary nodules in the mamma. He also asked Dr Stiles if he could explain what seemed to be a clinical fact, *i.e.*, that secondary deposits of cancer seemed often to pass through lymphatic ducts and only cause recurrent growths in the lymphatic glands beyond.

Secondary growths often attacked the lymphatic glands only, and yet the cells causing them must have come along the lymphatic ducts from the primary cancer.

Dr Bruce, in speaking to the pathological questions raised by Dr Stiles, referred to the new departure he had made in regarding carcinoma of the mamma as purely local and limited in its origin, and not as the result of a general cancerous transformation in the gland. He hoped that Dr Stiles would soon be in a position to demonstrate more fully the grounds on which he based this statement, as well as the evidence that the spreading carcinoma produced atrophy in the neighbouring portion of the acini of the mamma.

Dr W. Russell complimented Mr Stiles on the beauty and excellence of his demonstration, and considered the communication would be of much value if it led the surgeons to remove more freely than seemed to be their custom malignant growths, and from what had been already said that evening it appeared that this had already borne fruit. For himself he had long considered that in operating for the removal of malignant growths the consideration ought not to be how little could be removed, but how much it was possible and practicable to remove, including fat and connective tissues. With reference to the channels by which the disease spread, there could be no doubt that the lymphatics played an important part, and some of Mr Stiles' specimens showed well the mode of invasion in fat. As to the necessity for removal of the entire breast, it seemed to him that, theoretically at least, this did not cover the whole ground. The entire gland was not necessarily affected in cancer, and the whole gland might be removed, and yet secondary foci be left in neighbouring tissues. He differed from Mr Stiles on the question of spermatic influence; he (Dr Russell) was disposed to hold to the spermatic influence; he had specimens which appeared to show unmistakably the participation of the endothelium of bloodvessels in the malignant process. As to the so-called pre-cancerous stage, he understood thereby a deviation from normal, which, however, had not advanced sufficiently to lead the change to be recognised as malignancy, but this did not to him imply that the process was any less a malignant one. Mr Stiles himself acknowledged that this pre-cancerous stage could not be distinguished anatomically from the changes resulting from inflammation, and yet there could be no doubt that the processes were different; upon what element or factor this depended had yet to be determined, and he hoped Mr Stiles would continue his interesting and important work.

Dr Lovell Gulland expressed unqualified admiration for Dr Stiles' paper. He was specially interested in the fatty lymphatic glands of the axilla, and had carefully gone over Dr Stiles' specimens on previous occasions, with a special view to the question of their nature, and he was able to agree with Dr Stiles' view of their

character and origin. He could not accept Dr Russell's opinion that endothelial cells might actually become carcinomatous as the cancer spread, and thought that any appearance suggesting this should be examined very carefully.

Dr Barrett said he had listened with great pleasure to Mr Stiles' admirable paper; he wished to call attention, nevertheless, to the arrangement of epithelial cells in cystic tumours—namely, a tendency on the part of the epithelial cells to arrange themselves into spaces which more or less closely resemble acini or ducts of embryonic or nearly normal shape, and that this arrangement may show itself in lymphatic ducts. Also called attention to the very similar appearances met with in the cells of proliferated lymphatic endothelium, and this may in part account for the great cellular plugging seen in many lymphatic trunks both near to and at a considerable distance from the tumour.

Mr Stiles thanked the Society for the indulgent way in which his demonstration had been received, and for the generous remarks of the various speakers. He was glad to hear that a surgeon of the President's experience agreed with the principles of treatment which had been deduced from a study of the pathological anatomy of the disease. The plan, long ago advocated and adopted by the President, by which the skin over a tumour occupying the periphery of the breast should be removed, was a most excellent one, and there was abundant pathological evidence to show that this method of dealing with the skin should always be practised in such cases. Mr Stiles considered that the "recurrent" nodule at the site of a suture in Mr Miller's case was a coincidence, and that the stitch was in no way the determining cause. In reply to Surgeon-Major Black, he said that caustics should not be used as long as a knife was at hand. In answer to Mr Cathcart's question, he pointed out that there was no constant relationship between the position of the main tumour and the situation of the locally disseminated nodules. He agreed with Dr Russell that in some cases it might be necessary to remove more than the breast, while in others a partial excision might suffice. Since, however, the surgeon has no means of ascertaining which cases might successfully be treated by partial excision, the rule should be to remove the whole breast in all cases. He could not confirm Dr Russell's observation that endothelium underwent a cancerous transformation. Before sitting down, Mr Stiles begged to be allowed to thank the Surgical Staff both of the Edinburgh Royal Infirmary and of Chalmers' Hospital, for their kindness in placing such a wealth of material at his disposal. He felt that the exceptional opportunities thus afforded had increased his responsibilities to-night. He had endeavoured to meet them by bringing his results before the Society in a way which might be of some value to the practical surgeon. He felt he would have failed in this endeavour were it not that he had been fortunate enough to obtain the co-operation of Mr Andrew Pringle, whose skill in

photomicrography is now so well known and valued by all scientific microscopists. All the lantern slides illustrating the microscopic appearances have been made from Mr Pringle's photomicrographs of Mr Stiles' preparations, and to that gentleman is due a large share of the credit for whatever interest the Society may have taken in the demonstration. Lastly, he wished to be allowed to express his thanks and gratitude to Professor Chiene, not only for the interest he showed in the work during its progress, but also for the generous way in which he furnished the Surgical Laboratory of the University, where the investigation was carried out, with the necessary apparatus.

Meeting IV.—January 20, 1892.

Mr Joseph Bell, *President, in the Chair.*

I. Election of Members.

Dr Simson C. Fowler, Juniper Green, and Dr Towers Smith, London, were duly elected Members of the Society.

II. Exhibition of Patients.

Dr Sloan showed two cases of empyæma.

III. Exhibition of Specimen.

Professor Struthers exhibited a specimen of rider's bone. This preparation, Dr Struthers said, was the one he had shown in London at the Anatomical Society of Great Britain and Ireland in November 1887. He thought it would be interesting to the members of this Society to see a preparation of so rare a condition. A case had been recorded by Mr Birkett (*Guy's Hospital Reports*, 1868), and one by Mr Bryant (*Practice of Surgery*, 4th edition, 1884), in both of which the condition appeared to have had its origin in some rupture in the region of the adductor muscles during violent action on horseback. For this preparation he, Dr Struthers, was indebted to his former pupil, Dr James Allan of Leeds, by whom the dissection as it now stands was made. It was from a man, aged 55. The bone of each side is about $1\frac{1}{2}$ inch in length, $\frac{3}{4}$ to 1 inch thick, and mostly triangular. Right bone articulated to a projecting platform at the angle of the pubes, by a diarthrodial joint with very irregular surface. This bone was felt to be movable from side to side during life. Left bone immovably attached to the pubes. The muscular attachments are still seen on the left side. Adductor longus tendon directly prolonged from the end of the bone, which is flattened towards the tendon; attached to inner surface, on to the point, the fascia lata; to inner posterior border,

fore part of gracilis; to outer side, inner part of pectineus; behind, a large part of adductor brevis. All these muscular attachments are of full size. But, curiously, in regard to the origin of the ossification in this case, the man would seem not to have been a rider. Dr Allan mentions that "he had been a foot soldier for twenty-one years, but I have no note of his having been much on horseback in any capacity."

IV. Original Communications.

1. THE SPINAL COLUMN IN THE INFANT.

By J. W. Ballantyne, M.D., F.R.C.P. Ed., F.R.S.E., Lecturer on Diseases of Infancy and Childhood, and on Midwifery and Gynæcology, School of Medicine, Edinburgh.

A study of the anatomical characters of the vertebral column in the infant makes it clear that the spine is then in a transition stage. It has lost some of the features which were peculiar to it in the fœtus, and it has not yet taken on all the characters which it possesses in adult life. It is, therefore, of vital importance to the pediatric physician and surgeon to ascertain these age-differences, and to bear them in mind when studying the causation of spinal disease in early life, and its treatment, prophylactic and curative.

The statements respecting the measurements, curves, ossification, etc., of the infant's spine which are found in this communication are chiefly based upon the study (by the frozen sectional method) of a $6\frac{1}{2}$, a $7\frac{1}{2}$, and an $8\frac{1}{2}$ months' fœtus, and of two full-time infants, and upon the examination of four skeletonized fœtuses and infants, and the dissection of several fresh specimens.

The Length of the Spine and of its Regions.—The great relative length of the vertebral column is very marked in the fœtus during the earlier months of intra-uterine life, when the limbs are little more than buds from the trunk. As the full term of pregnancy is approached this disproportionate length of the spine is modified; so that in the new-born infant the vertebral column bears very nearly the same proportion to the total height as in adult life, and this is due, not to the length of the lower limbs, but to the large size of the head. In a male fœtus (A), whose age was $6\frac{1}{2}$ months, the spine measured 14·1 cms., and the whole body from vertex to heel 34·5 cms. in length; in an $8\frac{1}{2}$ months' male fœtus (C) the corresponding measurements were 16 cms. and 43·5 cms.; in a full-time male infant (B) they were 23 cms. and 56 cms.; and in a large full-time female infant (D) they were 24 cms. and 58·5 cms. respectively. These measurements were all made in the case of frozen infants in whom the cartilaginous element in the spine was present. The measurements of skeletonized infants give results which, although not free from fallacy, were practically identical

with those above mentioned. Thus, in the case of four full-time, or nearly full-time fœtuses that had been skeletonized, the average length of the body was 42·75 cms., and that of the spine 15·95 cms. It may be concluded, therefore, that during the two last months of intra-uterine life the length of the body is about two and a half times that of the spine.

When the relative lengths of the various regions of the spine (cervical, dorsal, lumbar, and sacro-coccygeal) are inquired into, it is found that both the fœtus and the full-time infant exhibit differences when compared with the adult state and that found in children of various ages. In the case of a 6½ months' male fœtus (A) the cervical and lumbar regions were found to be practically equal in length (2·7 cms. and 2·8 cms.); in a 7 months' fœtus they were exactly equal (3 cms.); and in an 8½ months' fœtus (C) they were nearly the same (3·5 cms. and 3·3 cms.) In the case of full-time infants it was found that the lumbar region exceeded the cervical in length: thus, in two infants, a male and a female (B and D), the measurements of the cervical and lumbar regions were 4·4 and 5·4 cms., and 4·6 and 5·1 respectively; and in three skeletonized infants that were nearly full time the figures were 2·8 and 3·7, 2·5 and 3·7, and 2·5 and 3·5 cms. respectively. It was found, therefore, that as the fœtus neared the full term of intra-uterine life the lumbar region grew in length more quickly than the cervical, and that in the new-born infant the former region was distinctly longer than the latter. In the adult the lumbar is to the cervical region as 3 : 2. There is, therefore, a more rapid growth of the lumbar portion of the spine as compared with the neck in the two or three last months of fœtal life and in childhood. This conclusion differs in one detail from that arrived at by Aeby, Cunningham, Symington, and others.

Aeby, in his paper on the "Age Differences in the Human Spine" ("Die Altersverschiedenheiten der menschlichen Wirbelsäule," *Archiv f. Anat. u. Entwickelungsgeschichte*, 1879, pp. 78-138), states that the cervical and lumbar regions of the spine are practically equal in length in the new-born infant; Cunningham comes to a similar conclusion; and Symington, from the study of frozen sections of four full-time infants, finds that the cervical part of the spine is very nearly equal in length to the lumbar. My measurements would seem to show that in the later months of fœtal life the growth of the lumbar portion of the spine has already begun to surpass that of the cervical, so that at the time of birth these regions are not equal in length, but the lumbar is distinctly longer than the cervical.

It is needless to specify minutely the relationship to each other of the lengths of the other regions of the spine, but a glance at the following table will give a general idea of the whole subject:—

Age of Fœtus.	Total Height.	Length of Spine.	Length of			
			Cervical.	Dorsal.	Lumbar.	Sacro-coccygeal.
6½ months' male fœtus (frozen), A, . . .	34·5	14·1	2·7	5·8	2·8	2·8
7 months' fœtus (skeleton), (1), ...	39	13·5	3·0	5·5	3·0	2·0
8½ months' fœtus (frozen), C, ...	43·5	16	3·5	6·5	3·3	2·7
Full-time fœtus (skeleton), (2), ...	43	16·6	2·5	7·1	3·5	3·5
Full-time fœtus (skeleton), (3), ...	43	16·7	2·8	6·7	3·7	3·5
Full-time fœtus (skeleton), (4), ...	46	17	2·5	7·2	3·7	3·6
Full-time male fœtus (frozen), B, . . .	56	23	4·4	8·9	5·4	4·3
Full-time female fœtus (frozen), D, . . .	58·5	24	4·6	9·3	5·1	5·0

In the case of the frozen fœtuses I have calculated out the percentage lengths of the various regions of the spine with the following results:—

Age of Fœtus.	Percentage Length of			
	Cervical.	Dorsal.	Lumbar.	Sacro-coccygeal.
6½ months (A), . .	19·1	41·1	19·8	19·8
8½ „ (C), . .	21·8	40·6	20·6	16·8
Full-time (B), . .	19·1	38·6	23·4	18·6
Full-time (D), . . .	19·1	38·7	21·2	20·8

From this table it will be seen that both in the later months of fœtal life and at the full term the dorsal region of the spine is about twice as long as the cervical; that in the case of fœtuses of 6 or 8 months the cervical and lumbar regions are practically equal in length, there being slight variations, sometimes the lumbar, sometimes the cervical being the longer of the two; that in the full-time infant the lumbar region always exceeds the cervical in length; and that in both the fœtus and full-time infant the sacro-coccygeal region varies much both absolutely and when compared with the other regions. In the case of skeletonized fœtuses and infants the same general results are found; but the changes caused by the shrinking of the cartilaginous and fibrous structures of the spine prevent such measurements from being so reliable as those made upon frozen sections. The percentage lengths of the various regions in the skeletons are appended in tabular form:—

Age of Fœtus.	Percentage Length of			
	Cervical.	Dorsal.	Lumbar.	Sacro-coccygeal.
7 months (1), . . .	22·2	40·7	22·2	14·8
Full-time (2), . . .	15·0	42·7	21·0	21·0
„ (3), . . .	16·7	40·1	22·1	20·9
„ (4), . . .	14·7	42·3	21·7	21·1

The observations of Aeby, Rasenel, Dwight, Symington, and Cunningham, show that during childhood and youth the adult proportion between the length of the cervical and that of the lumbar region is being gradually approached (*v.* Dwight and Rotch, *Archives of Pediatrics*, viii. p. 161).

The Flexibility and Curves of the Spinal Column.—In the fœtus and young infant the vertebral column is very flexible. Every one who has handled a baby must have observed this fact. Dwight and Rotch (*Archives of Pediatrics*, viii. p. 161) have experimented upon the degree of flexibility, and from personal observation I can confirm their results. They found that if the abdominal wall was cut through, and the abdominal viscera removed, the infant's head could be bent back so as to touch the buttocks, and that if the head and limbs were taken away and the ribs divided, the atlas vertebra could be made to touch the coccyx. They also ascertained that the middle portion of the spine was the most flexible part, and that the column could with equal readiness be bent forwards and laterally. It is, however, unnecessary to mutilate the infant's body in order to demonstrate the flexibility of the spine, for in the case of an intact specimen the head and heels can be bent backwards till they touch. The great ease with which an infant's spine can be bent is due to peculiarities in the spine itself, as will be immediately pointed out; but it is also in great measure permitted by the weakness of muscular action at this early period of life. In fact, the great flexibility of the cervical region is very largely due to weak or inco-ordinated muscular action. In connexion with this matter, the method of articulation of the head to the vertebral column is worthy of study. The occipito-atlantoid joint is not one calculated to allow much movement; and when the infant's head is flexed or extended the occiput moves only to a slight extent upon the atlas vertebra, the principal movements occurring at the cervical articulations. Now, if an examination be made of the articulation between the condyles of the occiput and the lateral masses of the atlas, the reason of the small degree of mobility here possible is made manifest. The condyles of the occiput are nearly flat at birth, and there is hardly any trace of that convexity (antero-posterior and lateral) which exists in later life. The articular surfaces upon the atlas also are flatter than in the older child, and the degree of movement between them and the facets on the occiput must necessarily be limited. The occipital condyles measure from 1 to 1·3 cm. in length, and from ·5 to ·6 cm. in breadth, and lie chiefly upon the ex-occipitals, only a small part anteriorly belonging to the basi-occipital part of the bone. In later life the condyles become more curved, and are consequently better adapted for extensive movement. Cleland (*Anatomical Memoranda*, vol. i. p. 18) describes specially two changes in the form of the occipital condyles which occur after birth,—the one is the curving of the articular

surfaces, the other is the growth of a wedge of bone in front of each condyle, which serves the purpose of preserving the balance of the head whilst the face and fore part of the brain are increasing in a relatively greater degree than the cranium and posterior part of the brain.

With regard to the curves of the vertebral column in the fœtus at an early stage of development, there is one general bending with an anterior concavity; later the sacral promontory appears, and there arise two curves, one below and one above the promontory, each with its concavity directed forwards. In case A (the $6\frac{1}{2}$ months' fœtus) these two curves are well demonstrated; the upper is, as we should expect, the more marked (Plate I., A). In the case of the infant at birth, it is usually stated that there is a curve with anterior concavity in the dorsal region. Now, the making of frozen sections has demonstrated the fact, that the position in which the body is placed is a most important factor in the production or in the abolition of spinal curves both in the infant and in the adult, but more especially in the case of the former.

The differences in the character of the spinal curves in the case of infants that have been frozen in different positions are shown in Plate I. In case A (the $6\frac{1}{2}$ months' fœtus) the body was placed in the freezing mixture in the dorsal posture, the head was slightly flexed, and the lower limbs were markedly extended. It is found that, whilst the cervical part of the spine runs practically vertical, there is a well-marked dorsal curvature with anterior concavity. The extension of the lower limbs has caused a distinct forward projection of the lumbar and sacro-coccygeal portions of the vertebral column. This gives to the promontory of the sacrum an apparently great degree of prominence, for it is really the lower lumbar vertebræ that cause the projection. In case C (the $8\frac{1}{2}$ months' fœtus) the body was frozen in the dorsal posture, with the head flexed to a slightly greater degree than in case A, and with the thighs flexed, abducted, and rotated outwards to a small degree. It is found that in this case the whole spine is nearly vertical. There is a faintly marked sacral promontory, a very slight anterior concavity in the lumbar and lower dorsal regions, and a sacro-coccygeal anterior concavity. The upper part of the spine may be said to be vertical, with a slight inclination forwards in the cervical region (Plate I., C).

In case B, a full-time male infant, the head was fully flexed upon the chest during the freezing process; and the thighs were flexed upon the abdomen, abducted, and rotated slightly outwards. There is in this instance a marked cervical curve with its concavity forwards, and this is continuous with a similar curve in the upper dorsal region. In the lower dorsal and in the lumbar region the spine projects slightly forwards. The sacral promontory is not

pronounced. The usual sacro-coccygeal anterior concavity is present (Plate I., B).

In case D, a large full-time female infant, the body was placed in the freezing mixture in the genu-pectoral position, and in consequence of this a marked change in the character of the spinal curves has been produced. On account of the posture the sacral region formed the highest part of the spine, and from the sacral promontory the vertebral column ran in almost a straight line towards the infant's head, inclining at the same time towards the anterior aspect of the body. The indication of a cervical curve with anterior convexity is found in the region of the neck, and this is manifestly due to the extended position of the head during freezing. There is also a faint trace of an upper dorsal curve with anterior concavity. The sacro-coccygeal curve is well marked (Plate I., D).

From the study of these sections it is evident that in the spine of the infant there are no fixed curves, save that produced by the promontory of the sacrum and the backward inclination of that bone. The curves that are present in the frozen section of the vertebral column are due to the position and attitude of the body during freezing. The spine can be made practically straight by slightly flexing the head, and by partially flexing the thighs upon the abdomen, the body being in the dorsal posture. A curve with anterior concavity can be produced in the cervical and upper dorsal regions by strongly flexing the head. A lumbar curve with anterior convexity can be made by fully extending the lower limbs upon the trunk. A cervical curve with anterior convexity, which may also affect the upper dorsal region, may be produced by strongly extending the head, *e.g.*, when the body is in the genu-pectoral position. Abduction and outward rotation of the lower limbs would seem to cause a curve with convexity directed towards the front in the lower dorsal and in the lumbar region. The variability of the curves of the infantile spine is thus abundantly proved, and fixed curvatures do not appear till some time after birth. Balandin is of opinion that a cervical curve with anterior convexity begins to be formed at the third month of extra-uterine life, when the infant begins to sit up in the nurse's arms; but Symington thinks that the cervical curve is never consolidated, and states that it can, even in the adult, be obliterated by strong flexion of the head upon the chest. In the infant, at any rate, the cervical part of the spine may have an anterior convexity or concavity given to it by extending or flexing the head upon the thorax. A dorsal curvature with concavity forwards is usually found at birth; but, as has just been shown, this curve cannot be looked upon as constant, for it can be altered by changing the position of the infant; it is usually present, because the infant usually is found lying in an attitude of flexion. Balandin states that when

the child begins to walk a lumbar curve with anterior convexity appears, but that this is not consolidated till adult life; and this is no doubt the case. The lumbar curve, like the cervical and dorsal, may be straightened out during infancy. A sacro-coccygeal curve with its concavity directed forwards does exist at birth, and is permanent; but in exceptional cases the sacrum and coccyx are directed downwards and backwards in a straight line, and in these cases there is often an abnormality in development, shown by the presence of what is called the "post-anal dimple." The production and fixing of the spinal curves are probably due in great measure to muscular action.

The Ossification of the Spinal Column.—The infant's spine is light and flexible, because it is not completely ossified, because it has still a considerable quantity of cartilage entering into its composition. Its flexibility is due also to another cause, which has been mentioned already. Little need be said about the degree of ossification found in the spine at the time of birth, for my observations generally confirm the statements found in anatomical text-books. At birth there are three primary centres of ossification found in each vertebra,—one for the body, and two lateral centres for the arches and processes. It is usually stated that the lateral centres are not united posteriorly at birth; but in several specimens I found bony union posteriorly—in one or two cases extending even into the spinous processes. The lateral centres are separated from the central centre for the body of the vertebra by cartilage—the neuro-central suture.

Such is the general plan of spinal ossification, but in certain vertebræ there are differences. The atlas vertebra has its anterior arch cartilaginous at birth; and it has two centres, one for each lateral mass, which unite posteriorly about the third year of life. In the case of the axis vertebra there exist at birth one centre for the body and two lateral centres for the arch and processes; but there are also two primary lateral centres for the odontoid process, which are often found fused together in the full-time infant. No trace of an apical epiphysary centre for the tip of the odontoid process is found at birth. The body and odontoid process of the axis are separated by cartilage at birth; they are said to unite at the age of three years, but probably union is incomplete till a much later date. D. J. Cunningham (*Jour. of Anat. and Phys.*, vol. xx. p. 238) goes so far as to state that the cartilage between the body and odontoid process does not disappear till old age, and supposes that authors have been misled upon this point by the peripheral ossification.

At birth the five parts of the sacrum usually show one central and two lateral primary centres in each; but the coccyx is then, as a rule, entirely cartilaginous, the centre for the first coccygeal vertebra appearing shortly after birth.

At the fourth month of intra-uterine life, *the spinal cord* occupies the whole of the vertebral canal; but thereafter the cord grows less quickly than the canal, and is at the time of birth found to terminate at the level of the first lumbar vertebra. The part of the spinal canal lying below this vertebra is occupied by the leash of nerves and the filum terminale, which together constitute the cauda equina. The relation of the spinal cord to the vertebral canal in the new-born infant does not differ from that found in the adult, but in premature infants and in fœtuses the commencement of the cauda equina is at a lower level than in the new-born full-time infant and in the adult.

A few words may now be said with regard to the spinal column in an *anencephalic fœtus* (Plate I., E). The specimen, which was one of well-marked anencephalus in an eight months' fœtus, was frozen in the dorsal posture with the thighs half flexed upon the abdomen and the arms lying by the side; there was no abduction nor rotation outwards of the thighs. The whole of the dorsal, along with the upper part of the lumbar region, forms a curve with anterior concavity; the lower lumbar vertebræ form together with the first sacral a well-marked promontory, and there is the usual sacro-coccygeal curve. The cervical part of the spinal column presents an interesting abnormality. The spine in the region of the neck turns sharply backwards, forming a curve with anterior convexity, the result being that a line dropped vertically from the tip of the odontoid process passes posteriorly to every part of the spine. There was spina bifida in the cervical region in this case, the arches and spines of all the neck vertebræ being absent. The curious curving of the cervical region of the spine in this case serves to explain how it is that anencephalic fœtuses appear to have no neck. The cervical part of the spinal column is not small, but from its curvature the neck appears to be very short, and the face comes to be approximated to the sternum. From the approximation of chin to sternum, the skin is found to pass from the one to the other without dipping down between them. On dividing the skin the chin can be easily separated from the chest. This fact I have noted on several occasions when dissecting anencephalic fœtuses. The backward inclination of the cervical spine in these cases is probably due to the condition of spina bifida which exists, and also to the abnormal ossification of the basis cranii. The odontoid process in this specimen was in close contact with the basilar part of the occiput; there was no foramen magnum, and the anterior part of the atlas was in all probability fused with the odontoid process, giving to the latter its disproportionately large size.

The segments forming the spinal column were in this anencephalic fœtus unusually well ossified for the age of intra-uterine life attained. Of course, the cervical region offers an

exception to this statement as regards the arches and spines, which are absent in that part; but the rest of the spine shows a degree of ossification greater than that usually found in a full-time infant.

The length of the spine was 21 cms., and that of the whole body 38 cms. The disproportionately great length of the spine was manifestly due to the absence of the cranial vault. The length of the various regions were as follows:—cervical, 3·7 cms.; dorsal, 8·3 cms.; lumbar, 4·5 cms.; and sacro-coccygeal, 4·5 cms. The fact that the lumbar region is nearly 1 cm. longer than the cervical is another proof that the spine in this case, notwithstanding the condition of spina bifida, is well developed for the age of fœtal life to which the infant had attained.

One or two of the *general conclusions* to which a study of the spinal column in infancy leads us may now be mentioned:—

1. The total length of the body of the infant at birth is about two and a half times that of the spine. This is due not so much to the lower limbs, which are relatively short, but to the head, which is large at this time of life.

2. In the case of premature infants (six or eight months' fœtuses) the cervical and lumbar regions of the spine are practically equal in length; but in well-developed, full-time infants the lumbar part of the vertebral column is longer than the cervical, although not so much longer as it is in adult life, when the lumbar spine is to the cervical as 3 to 2. (In the infant the proportion is approximately as 5 to 4.)

3. In the infant the spine is very flexible, and this flexibility is due not only to the imperfectly ossified condition of its segments, but also to the weak muscular action at this age.

4. There are no fixed curves in the infant's spine save that caused by the slight projection of the sacral promontory; those that are seen in frozen sections are due to the position of the body during freezing, and vary with the changes which the position may undergo.

5. Whilst there are no *fixed* curves in the spinal column in the infant, a general curvation of the spine above the sacral promontory usually exists (as it did also in fœtal life), and this has an anterior concavity (kyphosis).

6. If the bones be unusually soft and the muscles weak (as in rickets), and also if the infant be encouraged to sit up at too early an age, this natural and temporary infantile kyphosis may become pathological and permanent. Under similar conditions other wrong curvatures of the spine may also be produced.

7. In the new-born infant the characters of the facets of the

occipito-atlantoid articulations are not such as to permit of safe and extensive movements.

Description of Plate.

Representations of the spine from frozen sections of various infants. The bodies of the vertebræ are the only parts shown.

A. Spinal Column of a 6½ months' male fœtus.
B. „ „ full-time male infant.
C. „ „ 8½ months' male fœtus.
D. „ „ full-time female infant.
E. „ „ 8 months' anencephalic fœtus (female).

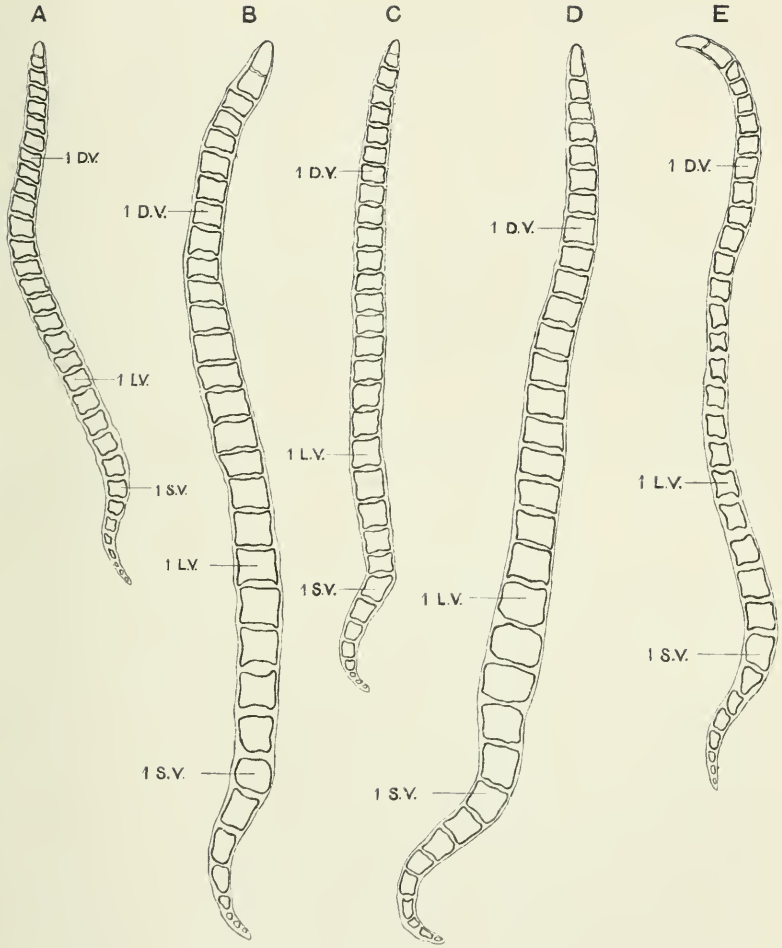
D.V. = Dorsal vertebra. L.V. = Lumbar vertebra. S.V. = Sacral vertebra.

The President said that the paper was an excellent continuation of the admirable series of original work already done by Dr Ballantyne, exhibiting as it did his characteristic determination to take nothing for granted, but work out every detail for himself. Amid his many conclusions, the President believed the fourth one to be of great surgical interest. In it he proves that the fœtal spinal column has no special curves, but will assume any curve according to the direction in which the body is placed. This bears in an interesting manner on the question of mechanical causation of spinal curvature in cases of rickets.

Dr Ireland, in answer to an inquiry from the President, said that he rarely had occasion to examine the spinal cords of idiots, but he believed that disease of the brain inducing idiocy was often accompanied by disease of the spinal cord. It was known that in microcephaly the spinal cord was generally smaller than usual as well as the brain. He had hoped that Dr Ballantyne in his instructive paper would have told them something about the lateral curvature to the right described by some anatomists as occurring in the adult, if not in the child, and regarded by some as the cause of right-handedness. He hoped that Dr Ballantyne might examine the question how the vertebræ grew, so that their cavity increased. Was it by absorption on one side and deposit on the other? The cranium was believed to grow by enlargement at the sutures.

Dr John Thomson was surprised that Dr Ballantyne should speak of the young infant's occipito-atloid joint as being more readily dislocated than that of adults. He thought that very few adults indeed could, with impunity, subject their necks to what those of many infants had to put up with in their passage into the world, and yet dislocation of the head during birth was fortunately not very common.

Dr Hughes asked Dr Ballantyne how he accounted for the diminution in the actual length of the cervical spine as traced



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in the list showing the lengths at the ages of 7th, 8th, and 9th months? In the 9th month fœtus the cervical spine was actually shorter than in the 7th month. Dr Hughes would like to know whether this was due to a flexed position of the head at the time of freezing.

Dr Ballantyne, in reply to certain questions that had been asked, said that he had not found any appreciable difference in the level at which the spinal cord terminated in male as compared with female infants. He had not been able to demonstrate any distinct lateral curve either to right or left in the infant's spine, and did not believe that any such curve then existed, although, of course, it could easily be produced by postural changes. With regard to the flattened character of the articular surfaces in the occipito-atlantoid joint, he believed that this might possibly explain the fatal accidents which sometimes occurred when children were lifted up by the ears, and he thought that if all infants that died during labour were carefully examined, it might be found that dislocation of this joint was not so rare as had been supposed. He had to thank the members who had spoken for their kindly and suggestive criticism.

2. AN EXTREME CASE OF HYSTERO-CATALEPSY—TRANCES LASTING 58, 30, 24, AND 12 HOURS; INSANITY; RECOVERY.

By Allen Thomson Sloan, M.D.

A. B., 19 years of age, pupil-teacher, of a sensitive and very affectionate disposition, religiously inclined, in ordinary matters and in the conduct of her class showing more than usual ability and self-possession; has three brothers and three sisters all perfectly healthy, with the exception of an elder sister, who suffers from anæmia. An aunt on the mother's side was subject to hysterical fits, and a sister of her father suffered from epilepsy. The father and mother were of distinctly alcoholic tendencies, while previous to her marriage the mother had lived rather a gay life. Menstruation began when she was 17 years of age, but has been regular, plentiful, and accompanied by no special pain further than slight headaches.

Two years ago—in September 1886—she fell down in the street, and was carried into a chemist's shop, where she remained unconscious for four hours. She was attended by a medical man for three or four days, but no definite opinion was given as to the cause.

The following autumn she was attended by me for a hysterical attack, which was followed by aphonia for three weeks. During the last week of January 1889 she took a fit in school, and was unconscious for four hours, and was seen by Dr Murray, who pronounced it catalepsy. For a few days afterwards she was peculiar

in manner, inclined to laughter and to make light of her attack. At this time she had a quarrel with the gentleman to whom she was engaged, and on the 31st of January went to a friend's house in the east end of the town, where she behaved in a very excited manner. She left about 8.30 P.M., after kissing her friend six times, coming back again and again to repeat her good-byes, and stating she intended to jump over the Dean Bridge. Several hours afterwards she was found sitting unconscious on the railings at Donaldson's Hospital, and was removed to Ward VI. Royal Infirmary, where she remained in that condition till between 9 and 10 A.M. Her mother questioned her, but all she could say was that she had felt her head light, and took a car home. She imagined she went to the tramway terminus, and wandered round and round some square under the impression she had asked her way home. Doubtless this lapse of memory immediately preceded a distinct attack of catalepsy. The fortnight after this she did not return to school, but was particularly bright and intelligent, and told all her hospital experiences. From inquiries, it seems that in Ward VI. it was considered at the time an ordinary hysterical attack.

On *Sunday the 24th of February* she lay in bed most of the forenoon, but rose and made her bed about 2 P.M., took a hearty dinner, and went to bed again about 4. Her mother noticed she took a slight convulsive fit, her face becoming purple and the breathing unnatural; she then turned on her side, and neither spoke nor moved in spite of all efforts to rouse her. She was seen first on Monday, 25th February, at 1 P.M., and was lying in bed with her hands stretched out at her sides, the body perfectly rigid, and to all appearance dead, save for the warmth to the touch, the bright colour in the cheeks and lips, and the faint but regular breathing occasionally broken in rhythm by an inaudible sigh, during which the chest was filled to its utmost capacity. The teeth and jaws were firmly clenched, the eyelids closed, but not tightly, the eyeballs turned straight up, the whites alone being visible, and the pupils with difficulty seen to be slightly dilated. The conjunctivæ were completely insensitive. Pulse was 90, well sustained, regular. The respirations 20 per minute, mostly natural, but occasionally broken by a prolonged inspiration and expiration about every fifth breath, but varying. The limbs were plastic, and capable of being moulded into any position, which, however peculiar, remained fixed. The head was bent on the chest, the back curved, each respective joint of the arm, the shoulder, elbow, wrist, fingers, the hip, the knee with difficulty was moved, and the result was the same, the ankle not so markedly as the others, and the toes remained flexible, but these only. Sensations of touch, heat, pain, and all the reflexes were completely abolished. Occasionally the saliva was swallowed; sometimes it frothed out from the lips. The jaw could only be opened and shut with great difficulty, and remained in whatever position it was put. Gravity had some effect in altering the posi-

tion, as when bent forward the patient very gradually sank back in bed, though the head kept flexed on the chest. The patient responded to none of the ordinary stimuli—shouting into the ear, repeated pin-sticking, pinching, and slapping, dashing of cold water over head, face, and neck, the application of smelling-salts to the nostrils, the blowing of snuff up the nose, the tickling of the throat by a feather, and pressure over the ovaries, though this produced a slight purple flushing of the face, as did also handling of the sensitive parts, which made one fancy unconsciousness was not altogether complete. Pins stuck into any part of the body produced no sensation of pain and no bleeding. The bladder was completely empty on percussion, the heart sounds distinct, unaccompanied by murmur, normal in strength, rhythm, and position. The only movement produced was on opening the eyelids, which caused continuous fibrillary twitching, more especially confined to the upper eyelids, and which remained for several minutes, or sometimes as long as she was under observation.

At 7 P.M. the condition was unchanged, except that there was external strabismus, and the breathing had become very shallow and rapid, sometimes as fast as 40 per minute, and broken occasionally and irregularly by a prolonged sigh. Efforts to rouse her again only resulted in a slight extra flushing of the face.

11 P.M.—Condition unchanged, but position of upper limbs unaltered from posture put in at previous visit.

Tuesday, 26th February, 9 A.M.—Limbs more rigid than ever; pulse, 90; respirations gentle, shallower, faster; bladder quite empty.

2 P.M.—Limbs slightly more mobile; conjunctivæ more sensitive; internal strabismus.

4.30 P.M.—Pulse, 96; respirations rapid, light, and shallow, as often as 90 per minute, every twenty-fifth being broken by a sigh, sometimes audible, and occasionally heard by the attendants in the room.

7 P.M.—Condition unchanged in any particular.

11 P.M.—Conjunctivæ quite sensitive; fibrillary twitching of eyelids well marked; muscles much less rigid than in the morning; respirations faint, rapid, 90 per minute; sighs more frequent and audible. Various rough efforts were again made to rouse her, and to banish any thoughts of malingering one medical friend moved a leg, while another fixed the arm, then the limbs were moved suddenly in the reverse directions. Douching, blowing snuff up the nostrils, and slapping from head to foot with wet towels were all again resorted to, the only effect being a purple flushing of the face, with flow of frothy saliva from the lips. Milk placed at the back of the mouth was partly swallowed, but mostly flowed back through the lips. Weights were applied to the various limbs; an ordinary kitchen iron was attached to the foot and arm while raised in the air, but they only swayed slightly, not greatly

lessening resistance, though time was not given to watch the action of gravity.

Wednesday, 27*th February*, 9 A.M.—Patient had remained in the same condition till 2 A.M. on Wednesday morning, having been in cataleptic trance from 4 P.M. on Sunday afternoon—fifty-eight hours—taking no food, responding to no external stimuli, with all her functions completely suspended, as the bladder continued quite empty, and no urine nor fæces were voided in bed.

At 10 P.M. on Tuesday a peculiar incident worth noting occurred. A brother was sent into the room to see how his sister was keeping, and thinking she looked very strange he fancied her dead, and leaning over her in horror, he cried out, "M——, are you dead?" She raised herself up, gazed vacantly and without recognition into his face, then sank stiffly and gradually back, her face flushing and her mouth frothing, in an apparently convulsive effort to speak. When wakened at 2 A.M., she asked her mother why she had allowed her to sleep so long, and was given milk to drink, which she swallowed quite readily along with some bread, and this was repeated two or three hours after. I saw her at 9 A.M., when she was lying smiling in a sort of vacant manner; and as her memory was quite confused and her mind disturbed, I thought it wiser to let her think she had just wakened out of sleep. The tongue was clean and moist, pulse 80, temperature normal, the muscles quite soft and flexible, and mobile at will. Her expression was absent and silly, and her thoughts carried her back to four weeks ago, to the seizure she had first taken, the last day she was at school. She complained of great muscular soreness all over the body, especially in the right arm, which had been most subjected to movement. At 11 A.M. she had some tea and fish, and at my suggestion got up and dressed, and went about the house as usual. She continued apparently well, but in a state of mental instability, easily excited to laughter or tears, till about 3 P.M., when I was suddenly sent for, to find her exactly in the same condition as before. The attack had come on quite suddenly in the kitchen, while sitting on a chair speaking to her father, who immediately tried to rouse her and divert her attention from herself. She spoke a little, saying her head felt very strange; and she wondered what was wrong, as she felt her jaw stiffening; then made an apparent unsuccessful effort to speak, and became unconscious, remaining fixed with her arms stretched out in the exact position she occupied in the chair. Her clothes were removed with difficulty, and she was forced into bed, where I saw her immediately afterwards. The limbs were again perfectly rigid, her eyes turned up in a state of external strabismus; pulse rapid, 120; and conjunctivæ completely insensitive. Suspecting possible malingering, I put her on the edge of the bed, with the shoulders hanging over and the head bent back, and poured a whole ewer of water over face, head, and neck, with no effect further than to produce the flushing of the face and

frothing at the mouth as before. I then extended one leg straight up from the hip, folded her two hands in an attitude of prayer, bent the head on the chest, the trunk leaning slightly forward, so that she was actually sitting on one hip on the edge of the bed, and in that position she remained without the slightest alteration, till her limbs were forcibly put in more natural positions. At 7 P.M. her condition was unchanged; but I learned that through the day she had passed a small quantity of urine, and the bowels had moved slightly, though unfortunately nothing was able to be preserved.

At 11 P.M., in her hearing, as I was under the belief she might still be shamming, I threatened to apply the actual cautery to the whole of her spine and to feed her by the stomach-pump. By previous directions, she had been left in the room entirely alone, being watched from a neighbouring bedroom, and a false alarm of fire was raised to rouse her, but in vain.

At 9 A.M. on Thursday the 28th she was lying on her left side, but still quite rigid. I turned her round, forced her into a sitting posture, with difficulty opened the mouth, and fed her with custard by the stomach-tube. Slight efforts at vomiting were made on the introduction of the tube, and her tongue moved slightly in the mouth, this act being followed by the flushing of the face and frothing at the lips as before. At 1 P.M. two large fly-blisters were applied over the ovaries. At 2 P.M. the conjunctivæ were more sensitive, and the muscles certainly less rigid than before. At 4 P.M. her pulse was 100, and the temperature in the axilla 99°, the first rise in temperature since the commencement of the attack. At 5 P.M. she was seen by Professor Annandale and his Resident, and for purposes of demonstration she was put in every conceivable peculiar position; her jaw was forced open, and she was again fed by the stomach-tube with beef-tea and egg, a small quantity of which came back by the mouth, and slight efforts at vomiting were made on the introduction of the tube, the face flushing and mouth frothing as before. Pins stuck in various parts of the arm produced no pain, and were followed by no bleeding, while the bladder still remained quite empty.

At 9 P.M. she had come out of the fit, having remained unconscious thirty hours. The muscles were quite mobile and responded to the will, but her pupils were widely dilated, and her memory was a complete blank as to what had happened during the last five weeks, while her smile was silly and vacant. She said her head felt light and dazed, just as if she were tipsy or had come out of chloroform, while her whole body felt stiff and sore. The pulse and temperature were normal. The sensation of touch was impaired in the fingers of both hands, and those of pain and heat delayed; the skin and deep reflexes were normal. During this second attack of thirty hours the urinary function was again suspended, as no urine was passed, and the bladder found quite

empty. The first urine passed was on Friday morning the 1st of March—quantity, 4 oz. of highly concentrated urine, muddy in colour, loaded with urates, acid in reaction, specific gravity 1026, containing a small quantity of phosphates, but no albumen nor sugar, and very slight traces of bile. The bowels moved once freely, but there was nothing of importance to note but that the fæces were dark in colour. On this (Friday) morning she was lying smiling vacantly, with pupils widely dilated and not readily responsive to light. The memory was quite confused and speech incoherent, though she complained of great muscular soreness, especially in the right shoulder and arm, and that her head felt strange and light. In this condition she remained all day, though in a high state of mental exaltation, easily excited, and inclined to become wildly hysterical and cry, and so on through the night, only being controlled by the firmness of her mother.

On *Saturday, March 2nd*, at 11 A.M., the pupils still continued much dilated, though less than on the previous day. Her manner was much more collected, and she was able to give a fairly definite account of her leaving school and what took place in it Wednesday five weeks ago.

On *Sunday*, at 10 A.M., the pupils were quite natural; her mental condition quite acute and reasonable; and the girl able to give a most intelligent account of her condition, and in every way quite well, except that her right arm from the shoulder downwards was rigid as before, remained in any position it was put, and was insensible to touch, heat, and pain. All the other parts were perfectly normal, except the plantar reflexes, which were much exaggerated, and the left hand and arm, which were cold and blue; the temperature 1° less than on the right side, distinctly sub-normal. The patient was sitting taking her breakfast quite calmly, and repeated her story of leaving school correctly and without hesitation.

At 6 P.M. patient's mind was perfectly clear and collected, but the right arm remained exactly in the same condition, when all the previous tests were re-applied, and, in addition, the faradic and galvanic currents all over the arm, and also to various parts of the spine, cervical and dorsal.

The arm was absolutely devoid of sensation, though both muscles and nerves responded to the strong faradic current, the flexor muscles especially being most responsive. The greatest effect was produced when the sponges were placed over the origin and insertion of the biceps muscle and over the commencement of the brachial plexus, also over the ulnar nerve at elbow and wrist. The galvanic affected individual muscles less, but when applied over the nerves moved the whole arm in successive jerks, but with the exception of slight tingling at the points of the fingers no sensation was felt, though the other hand could not hold the handles. The pupils were still widely dilated, the left

hand cold and blue, while on the under surface of the arm and elbow there was marked hypostatic congestion.

On *Monday, March 4th*, at 2 P.M., patient was exactly in the same condition as on the night previous, though complaining of a feeling of weight and deadness in the right arm. She was most anxious to have the battery, and was evidently desirous of getting well as soon as possible. The pupils were more natural and responsive to light. The purple colour of the lower part of the left arm more marked.

At 8.30 P.M. she was sitting sound asleep in a semi-upright posture with her supper half finished before her, her fork held between the bent fingers of the left hand, the right arm rigid as before. When wakened she seemed startled, but said she had an irresistible desire to sleep all day. The conjunctivæ were sensitive, and the temperature and respirations quite normal; as also was the urine, though the total quantity could not be accurately estimated.

On *Tuesday, 5th March*, at 11 A.M., patient was found lying on her left side soundly asleep, and was with difficulty roused, though even during sleep her arm was bent stiffly across her chest, as rigid in every particular as before. In the morning her father had found her asleep sitting over her breakfast, just as had been observed the night before. The pupils were again more dilated than natural. The battery was applied over the arm, along the spine, and over the ovaries. Applied over the cervical and dorsal regions, it had the effect of completely drawing back both shoulders; and over the ovaries it caused strong contractions of the abdominal muscles. Over all parts of the body painful sensations were produced, but on the right arm no sensation whatever, though the muscles freely responded to the current. The bowels had moved once freely, the motion being dark in colour, but very little urine continued to be passed.

In the evening, at 7.30 P.M., after having been fairly calm all day, but slightly nonsensical in her thoughts and suggestions, she relapsed into a general cataleptic condition, the unconsciousness and general rigidity being preceded by a flushing of the face, with fixing of the muscles of the jaw and slight frothing at the mouth as before.

At 10.30 P.M. she was found lying unconscious in bed, with her head lying well back on the pillow and slightly inclined to the left side, the balls of the eyes turned straight up with the pupils entirely hidden, and with difficulty found to be contracted. The conjunctivæ were almost insensitive, and movements of the eyelids produced fibrillary twitching, which lasted for a minute or two. The colour of the lips and cheeks was good, the pulse 90; respirations 40 per minute, mostly abdominal, faint and shallow, broken irregularly as before by a prolonged inspiration and expiration. All parts of the body, with the exception of the toes, were com-

pletely rigid, and the reflexes completely abolished. The galvanic battery was applied over various parts, especially over the left arm, all the muscles of which responded freely, particularly the flexors—the fingers, wrist, and elbow remaining fixed in any peculiar position the contraction of the muscles caused them to assume.

On *Wednesday, 6th March*, at 9 A.M., the condition was quite unchanged as regards sensation or rigidity. The eyes were in a state of external strabismus. The colour of the lips and cheeks was good, but the pulse very weak and rapid, 140 per minute, while respiration was very shallow and faint. She was again fed by the stomach-tube; and as her pulse was so feeble, half a glass of whisky was mixed with the egg and milk, after which the pulse improved slightly. Efforts of vomiting were made on the introduction of the tube, and about a tablespoonful of the egg flip came back through the lips. The tongue moved freely in the mouth, but the only general effect was flushing of the cheeks as before.

At 2.15 P.M. patient was again fed by the stomach-tube with beef-tea and egg. The pulse was much improved, 100 per minute, and the respirations, chiefly abdominal, 80 per minute. At 10 A.M. she had been lifted out of bed and laid on the floor, her muscles remaining quite fixed until raised into bed again, and she was still in a state of absolute unconsciousness. In the course of the afternoon the case was seen by Dr Byrom Bramwell, who for some days had been greatly interested in the account of it. At this time the patient was still completely cataleptic; and in addition to previous tests, together we laid her on the backs of two chairs, widely apart, and without any support whatever. There she remained, like some modern Galatea, bereft of life. Anxious to try the effect of drugs, Dr Bramwell first injected hypodermically half a grain of nitrite of sodium, with the effect of only slightly flushing the face; and it is very interesting to note here that the action of the drug was undoubtedly delayed. Ten minutes afterwards another half grain was injected, with the effect of producing a general blush, which spread widely over face and neck. No perceptible general relaxation of muscles was anywhere detected. After a short interval it was suggested we might try apomorphia, and this we did with some hesitation, as the pulse was far from good. 1-20th grain was first injected, but the only effect it had was to produce the deadly pallor which precedes sickness. The full dose, 1-10th grain, was then injected, and after a delay of twenty minutes, carefully timed, profound sickness was induced, accompanied by death-like pallor of the face and upper part of the chest, profuse cold perspiration, and great lowering of the pulse. Efforts of vomiting were then made, but only a little mucus and some remains of the custard were rejected. Next to the complete suspension of the urinary function during the trance, this remarkable delay in the action of so potent a drug as apomorphia is worthy of special notice, as also the fact that even during

the very act of vomiting no actual relaxation of the muscles was anywhere observable, the patient remaining profoundly cataleptic. In the evening, at 7.30 P.M., about two hours after the apomorphia injection, patient had awakened out of the trance, had no recollection whatever of what had taken place immediately before, and was in every respect fairly well, except for the rigidity of the right arm, which was completely devoid of sensation as before.

Next morning, *Thursday the 7th March*, Dr Bramwell kindly came to see the case about 9 A.M. The zone of insensibility of the right arm and shoulder was carefully mapped out, and extended half-way down the shoulder-blade and under the axilla, and all the ordinary tests were again applied. It was thought advisable to examine the eyes with the ophthalmoscope. The left eye was perfectly normal, but as soon as the bright light was turned on the right eye the patient became cataleptic where she stood. The condition of trance lasted till 9 P.M.—about twelve hours—and the appearances were exactly similar to those already so fully described. On wakening up, however, her mental stability was totally gone. Her right arm, which remained rigid as before, "had been petrified at the petrifying well at Knaresborough." She was no longer 19 years of age, but an old maiden lady of 50, whose hair was gray, and who had just returned from an imaginary voyage round the world, having been nearly wrecked in a terrific storm in the Bay of Biscay. French was a much more polite language than English; and to show her familiarity with it she wrote with her left hand, in beautiful letters and in very elegant French, an exciting account of the scene on board ship during the storm and threatened shipwreck. For a day or two she was allowed the run of the house, was sent out walking carefully guarded, and during that time she took her food and slept well, amusing herself by playing tunes on the piano with her left hand, but the delusions remained fixed as before. Several times she was seen during sleep, but on no occasion did the right arm relax or resume its normal condition. With some reluctance, but for the sake of better treatment, she was certified as a lunatic and removed to Morningside Asylum, under Dr Clouston's care.

It is worthy of note that when the attendants came to remove her, she stooped down and laced her boots with the right hand, evidently under the influence of strong excitement.

To Dr Middlemass I am indebted for the following notes taken in the Asylum:—

"The morning after admission she said that her memory was much improved, and she had got rid of some of the delusions she had expressed on the previous day, notably in regard to her age. She distinctly remembered her condition of the day before, and stated that she fully believed the statements she then made, but could give no explanation of them.

"She improved considerably, but about three weeks after

admission she was found in the morning in a condition of trance. She was apparently sleeping, was not roused when spoken to, and took no interest in her surroundings. The muscles were plastic, but had not the waxy pliability of the true cataleptic state, there being a marked degree of resistance to movement on her part. Her limbs could be placed in any position, however, and remained in the attitude given to them. She apparently felt no pain, took no food, and passed no water during the trance. The eyelids and fingers were not rigid, but were in a condition of fine rhythmical tremors. The temperature was 97° F., and the respirations were short, sharp, and rapid, with occasional paroxysms of sighing and deep inspiration. The pulse was slow, but regular. On everting the eyelids she turned her eyes up and rolled them about. She remained in the condition of trance for thirty-four hours, when she awoke quite suddenly. It was found that she had bitten her lip, which was bleeding slightly. She asked various questions as to what day it was, etc.

"Two days later she was found in the early morning in a similar condition of trance. This lasted till the same afternoon, when she again bit her lip and tried to make a fuss about the bleeding. Snuff had no effect in making her sneeze. When awake she asked if she had had the battery, and seemed desirous of attracting attention to herself. She was told to do some work, was given a pill containing valerianate of zinc and soda. The urine was found to contain oxalates and phosphates. She was discharged recovered after two months in the Asylum."

I had the satisfaction of hearing, the summer before last, that the patient had continued quite well, and was teaching in a school in Manchester.

The President remarked on the extreme care and accuracy with which this most interesting case had been detailed, and briefly gave the history of a case of catalepsy he had lately seen. A young lady from whom he had excised a cystic tumour of mamma made a good recovery. Some months later he was asked to see her, as she was supposed to be paralyzed and dying. He found her lying on her back, apparently unconscious, with the whole left side of her body in the state of spastic contraction associated with catalepsy. It was apparently absolutely anæsthetic, as deep insertion of pins and other stimuli induced no expression of pain. Having read accounts of the metallo-therapeutic methods of Charcot and Brocq, and being by a lucky accident the possessor of a sovereign, he pressed the sovereign firmly over the ovarian region for a few seconds, and then plunged the needle again into the blanched portion of skin. The patient started up with a yell of pain, and there was no return of catalepsy or anæsthesia.

Dr Hunter desired first of all to express his appreciation of the extreme care and marked ability with which Dr Sloan's case had

been reported to the Society. He recalled a somewhat similar one, though in a much less degree, where a young lady patient fell asleep during her meals, and remained in a trance-like condition, from which she could with difficulty be roused. Pressure over one or both ovaries was always successful in waking her up. In this colder climate and less excitable people, cases such as those described by Charcot are not frequently encountered, and therefore wide experience of their natural history is not to be expected. Regarding Dr Sloan's patient, they must all agree that he fully exhausted all the ordeals to prove that the case was not one of malingering; and if error was committed at all, it was rather in overdoing than omitting any of the tests which are usually had recourse to in such cases. With such manifest involvement of motor, sensory, and secretory nerve centres, it would be difficult to regard the patient as merely hysterical, whatever that vague term may mean pathologically; and the after-history of the patient went far to confirm this view of the case.

Dr Taylor said he had not, nor need any one have, any hesitation in rising at the call of the President, at least to express admiration for the excellent and thorough nature of the paper. Unfortunately, or perhaps fortunately—for such was his real feeling—he had not had any such case under his personal treatment, nor had he had occasion to study it. He thought that probably Dr Sloan from his present viewpoint agreed with some in thinking the case over-diagnosed and certainly over-treated. By the time a man writes a paper on a case his views become considerably enlarged and altered. It was therefore very candid in Dr Sloan to describe these with so much—almost confessional minuteness. Where true candour in these matters was found, it was to be appreciated.

Dr Gillespie was doubtful if the case was true catalepsy, the muscles being rigid and with tremors, and he could not agree with Dr Hunter in saying that it probably was not hysteria; for though under severe tests—which, however, she could know to be tests—she did not respond, under the excitement of a sojourn in an asylum, the right arm, hitherto powerless, became movable, and she could lace her boot. After apomorphia, too, the abdominal muscles acted. He was reminded of a case of catalepsy in a man who was alcoholic, but who had also anæsthesia in the cataleptic parts, and the sort of aphasia often called "barrel organism," in which he answered the first question correctly, but gave the same answer to the next ten or so. Perhaps Dr Sloan's patient was alcoholic. He also related a similar case cured by Mendel in Berlin by the apposition to the part of a magnet.

Dr Ireland said that Dr Sloan's case was analogous to the form of insanity generally called *melancholia attonita*, or melancholy with stupor. It did not seem to him to be connected with hysteria, and occurred in men as well as in women. Such diseases, however, had

a great variety of symptoms. Probably in this case, along with paralysis of the motor centres, there was remaining a dim and vague consciousness. This was shown by the traces of emotion noticed when she heard them speak of her death. Patients after recovering from trance sometimes recalled that they had felt great distress on hearing those around say that they were dead or dying, although they were at the time powerless to move or to utter a word. The value of the case consisted in the very careful observation of the different symptoms. Improvement in our knowledge of insanity was in great measure dependent upon the studies of physicians who had opportunities of seeing mental derangement in the nascent state. Probably the paralyses of one or other side were dependent upon sudden changes in the distribution of blood to different portions of the brain or to one hemisphere.

Dr Craig remarked that, while he had no experience of such cases, yet he admired the admirable and lucid way in which Dr Sloan had narrated this interesting case. He believed with Dr Hunter and others that there were important changes in the nerve centres, and that this condition was aggravated to some extent by the kidneys ceasing to secrete urine during these attacks. With regard to the use of drugs for overcoming the characteristic muscular rigidity in these cases, he suggested the inhalation of chloroform, one of the most powerful substances known for overcoming muscular rigidity, believing that when pulse and temperature were normal it could not do harm, and in all likelihood would do good.

Dr Sloan, in reply, stated that should he ever meet with a similar case again he would feel disposed to try the effects of the remedy so effectually used by the President. It was his opinion from the first that this was no mere case of malingering; but such cases as these were in Scotland extremely rare, and in spite of all the ordeals applied some physicians seemed still to fancy the condition might be simulated. Dr Gillespie had suggested that the case was not one of true catalepsy; but rigidity of a plastic nature associated with fibrillary tremors is characteristic of that disease, and catalepsy seems to be an extreme form of hysteria. The title of the paper, Hystero-Catalepsy, itself explained the view taken of the case. The patient was not alcoholic, though she was of distinctly neuropathic constitution, and her heredity such as is known to exist in similar cases—alcoholism and immorality on both sides. The pathology of catalepsy was quite unknown. There was no paralysis of the motor centres, rather a loss of inhibitory power over the motor centres, or an actual stimulation of them, the muscles during the trance remaining in a state of constant tonicity. Dr Craig thought the arrested function of the kidneys had something to do with the attacks; but the patient was in a condition of absolute trance, and there seemed complete cessation of excretory functions, while the urine when passed was practically normal. Dr Sloan would not feel inclined to try the effects of chloroform

in such a case. The effects of the apomorphia were serious enough, and it would be with the greatest hesitation he would try such a powerful remedy again. He begged to thank the Society for their more than kind reception of his paper.

Meeting V.—February 3, 1892.

Mr Joseph Bell, *President, in the Chair.*

I. Admission of Ordinary Members.

Dr Alexander Miles, 25 St Bernard's Crescent, and Dr Robert Abernethy, 18 Alva Street, were elected Ordinary Members of the Society.

II. Exhibition of Patients.

1. *Dr W. Stewart* showed a case in which the ULNAR NERVE was divided by a heavy weight falling on the arm while the palmar aspect of the arm was resting on a sharp edge. The nerve was not exposed at the bottom of the wound, which united by first intention. A fortnight after an exquisitely painful spot developed above the cicatrix, and all the muscles supplied by the ulnar nerve below the wound became paralyzed and atrophied; the sensibility of the skin supplied by the ulnar nerve became almost, but not entirely lost, and ultimately the "claw hand" was well developed. The patient tried his work, but was unable, on account of the loss of power and sensation, to continue at it. After a trial of blistering and electricity, the nerve was exposed—six weeks after the injury. It was found to be flattened, involved in cicatricial tissue, and on the central end a traumatic neuroma had formed, showing that the nerve was completely divided, though the sheath of the nerve was not. This neuroma, involving $\frac{3}{8}$ of an inch of the nerve, was removed, and the nerve sutured; the hand was then strongly flexed and fixed in this position. Sensibility returned in about a week or ten days, and the patient was able to resume his work as an engineer on board ship in three months, and now, three years afterwards, has a hand which for his purposes is practically perfect in appearance, motion, and sensation, excepting an increased sensibility to cold in the little finger and ulnar side of the hand.

2. *Dr John Thomson* showed a child with MARKED PULSATION IN THE EPISTERNAL NOTCH, apparently due to the left carotid artery passing obliquely across that region from right to left. The case was of interest in connexion with tracheotomy.

3. *Dr Stockman* showed for *Dr R. W. Philip* a boy $4\frac{1}{2}$ years old, who was brought to the Royal Infirmary a fortnight ago, suffering from FACIAL PARALYSIS. There was no history of injury or exposure

to cold, and there was no ear disease. The probable cause of the paralysis was the pressure of enlarged indurated glands which were present at the angle of the jaw, and could be felt by the finger introduced into the mouth as a firm mass, tender to pressure. Such a cause is mentioned by Henoch, but is stated by him to be uncommon. The child had also large mammæ, the enlargement having been present since his birth, and consisting of adipose tissue.

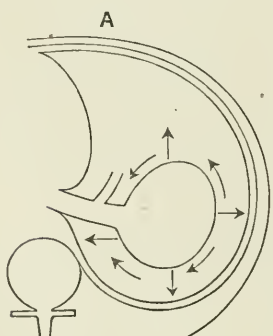
III. Original Communication.

EMPYEMA, BASAL LUNG DISEASE, AND BRONCHIECTATIC CAVITIES.

By Alex. James, M.D., F.R.C.P. Ed., Assistant Physician, Royal Infirmary, and Lecturer on the Practice of Physic, School of Medicine, Edinburgh.

That bronchiectatic cavities are frequently the result of too long existent pleuritic effusions every one will admit. The lung being bound down by a layer of fibrinous membrane, and the fluid being gradually absorbed, the diaphragm will be drawn up, the heart and opposite lung drawn over, and the chest wall drawn in. This drawing up, in, and over will not, however, in bad cases be sufficient to fill up the space which the absorbing fluid tends to leave, nor will in addition be the emphysematous distension of the affected lung at parts still pervious to air. Hence the bronchial tubes, the most yielding parts, will have their walls drawn out, and bronchiectasis will be produced. But what I wish to contend for in this paper is that a purulent pleuritic exudation—that is to say, an empyema—is, by its being able much more readily than a serofibrinous effusion to burrow through the lung tissue and open into a bronchial tube, a quite as frequent cause of basal lung disease and bronchiectatic cavities.

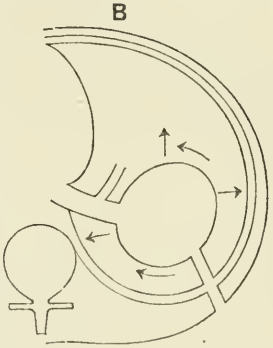
To explain this, let us consider some points as regards the physics of the lungs and chest wall.

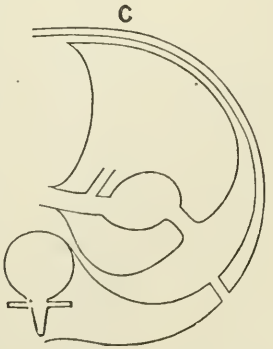


Suppose we have a bronchiectatic basal cavity at (A), the forces which tend to keep this cavity enlarging are, —(1), acts of coughing; (2), pressure of secretion; (3), traction, as the result of cell infiltration, fibrosis, and contraction in the lung tissue around. As regards 1 and 2 nothing more need be said; it is evident that both must always act in distending the cavity. As regards (3), the traction, we have to notice that it will act partly in distending and partly in contracting the cavity. Thus, as the newly-formed fibrous tissue contracts in every direction, it will, as shown by the straight arrows (Fig. A), by drawing the cavity

wall to the parietes, have an enlarging effect. On the other hand, by its contracting in the opposite direction, as shown by the curved arrows, it will at the same time act in contracting the cavity. Which of these effects of this fibrotic contraction will be the more marked? With a very large cavity—that is to say, a cavity nearly as large as the pleural cavity itself —the enlarging effect would certainly be the greater; but with most smaller cavities, having in consequence of their smaller size a considerable amount of lung tissue around which can yield to some extent, the contracting effect will be the greater. In ordinary large cavities, however, there will be very little difference in favour of the latter; so that, as against the distending effects of the coughing and of the accumulating secretion, any such contraction of the cavity will have little chance of occurring.

It is quite otherwise, however, when the cavity is opened and drained through the chest wall, as at (B). Here the secretion cannot accumulate, and so can have no distending effect, whilst the cough, owing to the drainage opening through the chest wall, may as a distending force practically be neglected. The contracting effects of the fibrosis will now have a chance of manifesting themselves, the walls of the opening through the lung tissue and the adhesions between the pleura and ribs will both be drawn upon, so that with a diminution in the size of the pulmonary cavity more and more space will be yielded by the pleural cavity, as shown in Fig. C. This was precisely what happened in the case of a man, G. W., who was admitted to Dr Muirhead's wards in the Royal Infirmary on the 11th August 1887. He had the symptoms and physical signs of a very large cavity in the lower part of his right lung, and on the night of his admission he brought up about a pint of very fœtid muco-purulent fluid.





Three days after admission the cavity was opened, and drained in the usual way. The opening in the chest wall was made a little outside of the line of the angle of the right scapula, and the huge cavity was easily struck some two inches within the pleura, the pleura and lung being firmly adherent all round. The patient was immensely benefited by the operation, but the discharge never

entirely ceased, so that drainage had to be continued. Becoming more and more exhausted, he died in April 1888. On post-mortem examination the following were the appearances met with:—

"Right lung weighed 1 lb. 7 oz. This lung was adherent a little way from its anterior border to the chest wall by a ridge of dense, hard, thick adhesions, which cut almost like cartilage. On cutting through this ridge, and thus separating the lung from the thoracic wall, a large cavity was opened into which was evidently the pleural cavity. The lung itself was collapsed toward the spine, and was covered by a dense tough membrane, the thickened visceral pleura. The lung retained its connexion with the parietes across the cavity by means of two thick pillar-like structures about the thickness of a florin; these were evidently limited areas of pleural adhesions which had been formed before the lung collapsed as a whole. The central part of their pillar-like structures contained pigmented pulmonary tissue, in which the alveoli were clearly visible to the naked eye. These two structures were situated on the upper part of the collapsed lung.

"On the surface of the lung, on its posterior aspect and near its inferior border, there were two openings the size of a large goose-quill; these were at the bottom of funnel-shaped depressions, and were quite evidently of long standing. One of these opened into a small cavity the size of a hazel nut, which had fibrous walls. This cavity was in connexion with a somewhat dilated bronchus. The second one was in connexion with an infractuous cavity, which ran for about three inches upwards in the substance of the lung. This cavity had its walls formed of pigmented alveolar tissue, and presented no fibroid thickening, as old cavities do; the largest part of this cavity would hardly hold a pigeon's egg—the greatest part of it was not one-third of that size. In the apex the tissue of the lung was tunnelled with cavities which communicated with one another, and presented the further interesting fact that they were in connexion, by long passages, with the pulmonary tissue in the pillar-like structures already referred to. Sections of this lung presented many different stages of destructive action. The more unaffected portions were deeply pigmented and leathery. The pleura was very much thickened, the bronchi were of deep purple colour, and tubercles were visible in their mucous surfaces.

"The left lung weighed 3 lbs. 1 oz. It was fixed by old but not very dense adhesions. The lung looked large and bulky; it had many hard nodules and masses of various sizes in its substance and projecting on the pleura. At the apex there was a recent cavity the size of a pigeon's egg, and round it was an area infiltrated with yellow tubercles. In the upper part of the lower lobe there was a considerable area densely infiltrated with gray and yellow tubercles. All the nodules found in the lung were found on section to present the appearance of gray and yellow tubercles. The changes in this lung were most marked and older in its anterior

third than in its posterior two-thirds. Adjoining the pericardium, for instance, a considerable area of lung was honeycombed with cavities, varying in size from a large pea to a hazel nut."

In this case the pulmonary cavity was evidently so large that complete cicatrization could not possibly occur; but the point which I want to emphasize is that, as stated above, the diminution in size of the pulmonary cavity meant the formation of a pleural one. Now, my contention is that similarly a collection of pus in the pleural cavity will, if allowed to drain through the lung, readily produce a pulmonary or bronchiectatic cavity.

In the case of an ordinary circumscribed empyema the lung tissue around is fibrosed from compression and irritative inflammation, and the acts of inspiration, expiration, and coughing, have little effect on the pus.

But suppose this pus ulcerates its way through the lung tissue into a bronchial tube? Its escape by the tube is not usually followed by entrance of air into the empyema cavity, and so there occurs an approximation of the pulmonary to the costal pleura, and consequent tendency to obliteration of the empyema cavity. The escape of pus will also, it is to be noted, be favoured by every inspiration, and the approximation of the pulmonary to the costal pleura by expiration and coughing.

In time, by this suction and force-pump-like action of inspiration, expiration, and coughing, the pus will be got rid of, the pleural membranes become again adherent, and in favourable cases this will mean the recovery of the patient. But the course is not always so favourable. The suppurative inflammation at the base of the lung caused by the burrowing of the pus is very likely to induce irritative inflammation and loss of tissue. This loss of tissue has to be made up for by fibrosis and contraction, which, as we have already seen, must act by drawing bronchial walls to pleura, and so tending either to separate again the pleural membranes or to dilate the bronchial tubes.

When we reflect that the act of coughing is all in favour of the dilating process, and that the irritation of the bronchial mucous membrane by the pus will cause increased secretion, and in time a similar distending tendency, we can, I think, understand how in this way, with obliteration of the pleural cavity, bronchiectatic or basal lung cavities are sooner or later likely to be produced.

Since ordinarily we do not see instances of basal cavities which we believe to have been produced by empyemas bursting through the lung until long after all traces of the empyema have disappeared, it is difficult in any given case to be sure that empyema has been the cause. The following is, however, I think, an example of such a case:—

H. P., a little girl of 9, was brought to me at the Infirmary in September 1889. The history was that she had been well till about two years previously, when, after exposure to cold, she had

"inflammation" in the right side. The nature of this inflammation could not clearly be ascertained, but she had had pain in the side, cough, fever, and sweatings. After some two months, during which time she was mostly in bed, she suddenly coughed up about a teacupful of fœtid purulent matter. She seems to have improved somewhat after this, but the cough and expectoration of fœtid matter, though in smaller quantity, continued, and she had remained weak and feverish, and was manifestly ill.

On examination, dulness on percussion and cavernous breathing and resonating crepitation were heard over the lower half of the right lung, and some harsh breathing with crepitation at the base of the left.

Becoming gradually weaker, and with the cough and fœtid spit persisting, the child died some six months afterwards. On post-mortem examination, the larger bronchi at the base of the right lung were found much dilated, and the surrounding lung tissue in a state of fibrosis. The pleura all over the lower lobe was adherent except at a spot laterally, where about an ounce of yellow serous fluid was found encapsuled. At the left base there existed, but to a very much less marked extent, a similar condition of lung and bronchi.

In this case there had been, I believe, a small, possibly fœtid empyema, which, bursting through the right lung, had sought to empty itself through a bronchus. In this it had succeeded to the extent of allowing pulmonary and parietal pleuræ to come in contact, but by its irritative and destructive effects on the lung tissue it had caused loss of tissue, fibrosis, and dilated tubes, this dilatation being favoured by the harassing cough which the infective purulent bronchitis necessitated. At first, and probably for months, the right side only had been affected, but latterly extension of mischief to the left lung had occurred, as the result, probably, of some of the fœtid purulent secretion finding its way to the bronchi at the base of the lung on that side. The small encapsuled pleural cavity containing clear fluid might have been either the remains of the original empyema cavity, or a cavity formed as the disease progressed by the traction of the fibrotic lung tissue on the pleura.

What I desire to draw attention to in this paper is that an empyema may act in this way, and be a cause of basal lung mischief, which is very likely to be fatal within a few months or years. I wish also to point out that such empyemas, often small and localized, are not at all uncommon, and often very difficult to diagnose, repeated exploratory punctures frequently failing, time after time, to reveal the purulent collection. Fortunately, however, repeated punctures are perfectly safe if properly carried out, so that practically there is little excuse for failure to recognise and treat such a condition.

In conclusion, I have to allude to the fact that an association

between empyema and purulent bronchitis or lung mischief has been noted by Sturges[1] and Drummond.[2] It seems to me, however, that the consideration of the physics of the lungs and pleura, such as has been entered into in this paper, is of very great value in enabling us to understand this association.

Surgeon-Major Black said that as diaphragmatic empyema had a tendency to evacuate itself upwards into the lung, he asked if Dr James would recommend any surgical procedure for the evacuation of the pus externally on the chest so as to prevent this tendency.

Dr James Ritchie said that his experience of the frequency of bronchiectasis did not accord with that of Dr James. He found bronchiectasis to be a very rare disease. Although some cases presented considerable difficulty in diagnosis, he found that by careful percussion and auscultation even small collections of pus could be located with tolerable exactness, unless they were very deeply seated, and that exploratory puncture, frequently repeated, was not required.

Dr Norman Walker asked Dr James how he suspected the condition of localized empyema which could not be diagnosed by physical sign? Did he finally find the empyema at the seat of his earlier tappings? He remarked that the repeated use of the same needle for several exploratory punctures was not to be considered a satisfactory amount of asepticism, and pleaded for the more free use of the dry heat sterilizer before making exploratory punctures.

Meeting VI.—March 2, 1892.

Mr Joseph Bell, *President, in the Chair.*

I. Election of Members.

Dr Robert Dundas Helme, 8 Brunswick Street, and Frederick Thomas Anderson, L.R.C.P. & S. Ed., L.F.P.S. Glas., 20 Inverleith Row, were elected Ordinary Members of the Society.

II. Exhibition of Specimens.

Dr William Russell showed a series of pathological specimens.

III. Exhibition of Patient.

Dr John Macpherson showed a patient exemplifying the result of treatment in myxœdema, and read the following notes regarding the case:—

The history of our knowledge of myxœdema, based upon the

[1] *Medical Press*, July 1, 1891.
[2] *Brit. Med. Journal*, July 14, 1891.

experimental researches of Mr Victor Horsley in this country, and of Dr von Eiselsberg and Professor Kocher on the Continent, is so well known that it is unnecessary for me to detail it. The chief English literature of the subject is to be found in Mr Horsley's Brown Lectures, 1885, the Report of the Clinical Society of London in 1888, and Dr Byrom Bramwell's *Atlas of Clinical Medicine*, vol. i., 1891. Since then some experimental clinical cases have been recorded relating the effect of grafting the thyroid gland of a sheep upon myxœdematous subjects. The chief of these was published in *La Semaine Médicale* for 13th August 1890 by two physicians of Lisbon—Drs Bettencourt and Serrano.

In the *British Medical Journal* for 10th October 1891 there are two independent articles—the first by Dr Murray of Newcastle, and the second by Mr Fenwick of the London Hospital—both describing the result of the hypodermic injection of a prepared extract of the thyroid gland in cases of myxœdema. The authors of these articles, separately and independently, reason that as the beneficial result of the grafting of the gland is so immediate, it must be due, not to the vascularisation of the gland tissue in the human body, but to the rapid assimilation of the free glandular secretion.

That their reasoning was correct is shown by the satisfactory results obtained in both instances by the hypodermic injection of the gland extract.

In the case which I now present, the same almost instantaneous relief of some of the most pronounced symptoms was manifest soon after the operation, and this speedy effect of treatment was, I have no doubt, due, as in other cases, to the absorption of the secretion of the thyroid gland. But I have also reason to believe that a considerable portion of the gland I grafted became ultimately vascularised in the body of the patient; and I cannot help coming to the conclusion that where there is the possibility of the successful vascularisation of the gland tissue after grafting, the latter operation should be much preferable to that of injecting the juice of the gland which, in the absence of direct proof to the contrary, would seem to be merely a palliative remedy.

The family history of my patient is unimportant, with this exception, that her brother was born a deaf-mute.

The patient is 39 years of age, and at the time of the first onset of her present symptoms she was 36 years old. She came under my care first in May 1890. She was then labouring under melancholia, with a distinct tendency to stupor. Her manner was listless and apathetic, and she could with difficulty be roused from her lethargy. She refused food, and had to be forcibly fed. Her pupils were equal, and reacted sluggishly to light and accommodation, but the tendon reflexes, on the other hand, were quick and exaggerated. Her temperature in the axilla was sub-normal, and her extremities were blue, cold, and swollen. Speech was thick, drawling, and

deliberate. Movement and co-ordination were slow and clumsy. She gradually recovered without manifesting any symptoms worthy of note, and was discharged from the asylum within three months of admission.

She was again admitted on the 22nd August 1891, having been transferred from the Edinburgh Royal Asylum, where she was a patient for ten days. She manifested the same mental symptoms —melancholia with stupor, and in addition the following nervous symptoms:—

1. Delusions of fear. She used to lie awake all night in terror of some impending calamity.
2. Vaso-motor disturbance. Her extremities (hands, feet, tip of nose, lobes of ears) were cold, swollen, blue.
3. Lethargy, with a want of spontaneity and interest in her surroundings.
4. Refusal of food.
5. Clumsy, slow movements.
6. Exaggerated tendon reflex.
7. Well-marked vertical headache.

In addition, she presented the following symptoms of myxœdema:—

1. Well-marked supra-clavicular swelling and filling up of the triangles of the neck.
2. Enlargement of the tongue, which was soft, flabby, and indented with the mark of the teeth.
3. Waxy appearance of the face, with the characteristic flush on the cheek.
4. Mal-nutrition of the skin and its appendages—hair, nails, etc. The hands presented the usual spade-like appearance.
5. Anæmia. This symptom was a prominent one, and the ocular conjunctivæ had the appearance of parchment.
6. Menorrhagia. She menstruated once a fortnight, and was ill for a week at a time, thus menstruating for two whole weeks out of four.
7. The quantity of urine was considerably diminished.
8. The temperature in the axilla was subnormal.

Two months after admission the mental and physical condition of the patient not having improved, but, on the contrary, appearing to be worse, I resolved to perform the operation of grafting a portion of the thyroid gland of a sheep, after the suggestion of Mr Horsley. The patient was accordingly placed in bed for a week previously, so as to be more closely observed. She was kept under the most favourable conditions as regards warmth and diet, and accurate observations were made as to her condition. During the seven days prior to operation the average daily temperature in the axilla was 97°·2, and the average daily amount of urine passed was 29 ounces. The urine contained no sugar and no albumen.

On the 22nd of October last the following operative procedure was undertaken:—

The right lobe of a sheep's thyroid was taken from a sheep then being slaughtered at the Asylum farm. The utmost antiseptic precautions possible were observed in removing the gland, which after the removal was washed in a warm weak solution of perchloride of mercury. The gland, which was removed by my assistant, was carefully protected in transit from the farm to the Asylum in a warmed glass jar. In the meantime I had the patient anæsthetized, and both infra-mammary regions were carefully washed and disinfected. The lobe of the thyroid was divided transversely into two equal parts, and each half was then split longitudinally. Two half-curved incisions were made in the infra-mammary region of the patient through skin and subcutaneous fat. A portion of the excessive fat was removed, and a piece of gland 1½ inch long was placed in each wound in such a way that the cut surfaces of the split gland were in contact with the raw surfaces of the flaps of the wounds. The wounds were then closed and dressed in the usual manner. The wounds did not heal by first intention, and there was considerable suppuration afterwards. If I might presume to suggest some points in regard to the surgery of the operation, I would say—

1. That the method of slaughtering sheep usually employed makes it extremely difficult to remove the gland with ordinary cleanliness, to say nothing of absolute asepsis.

2. That bleeding the animal to death necessarily depletes the vessels of the gland itself to such an extent as to make it doubtful whether vascular connexions can ever successfully take place in the human body.

3. On account of the redundancy of fatty tissue in the infra-mammary region, I do not consider it a suitable part of the body for grafting tissue like the thyroid gland. I think a more vascular, more muscular locality should be selected.

The results of the operation were as follows:—

1. Within twelve hours there was such a marked mental improvement in the patient as to be noticeable by every person who came into contact with her. She became talkative, cheerful, answered questions readily, and the mental reflex, which was formerly so slow, became altered and quickened. Her intelligence and spontaneity markedly increased. Her speech, however, remained slow and drawling.

2. The average daily temperature for seven days after operation was 99°·2, and for nineteen days afterwards 98°·9.

3. The average daily quantity of urine passed for nineteen days subsequent to the operation was 41 ounces.

4. The fear at night, the melancholia, and delusions disappeared on the day following the operation, and have not since recurred.

5. The vertical headache more or less persistent for three years has not since been felt.

6. The anæmic condition has been removed.

7. The skin is now soft and smooth, and the hair is not so dry.

8. The quantity of urine passed for the twenty-four hours ending on the 28th day of February was 52 ounces.

9. Menstruation, which had for three years previously been irregular in the manner described, is now of monthly occurrence, and lasts only three days.

In conclusion, I do not claim that the operation has removed the myxœdematous condition, but it has relieved the symptoms, which, with the exception of the melancholia and stupor, have all been more or less constant for three years. Under favourable conditions, I see no reason why the relief should not continue. These favourable conditions are:—

1. Warmth, upon which Dr Byrom Bramwell insists in his very able and lucid article on Myxœdema in his *Atlas of Clinical Medicine*.

2. Farinaceous dietary, which I infer from Mr Victor Horsley's recent researches, as published in two papers in the *British Medical Journal* this year.

3. Avoidance of worry and over-work, which most authorities are agreed in recognising as a causative agency.

4. Perhaps massage over the gland, which Professor Grainger Stewart and Dr John Thompson have used with success in one case, might with advantage be practised.

Finally, I would remark with reference to the sudden mental and physical change following the operation, that I entirely agree with Dr Murray and Mr Fenwick in the opinion, expressed by them in the *British Medical Journal* of 10th October 1891, that the sudden change could not be due to the engrafted thyroid gland becoming functional, but to the absorption into the system of its special secretion.

Dr Lundie said he had a case of myxœdema at present under treatment by subcutaneous injection of extract of sheep's thyroid, according to the method of Dr Murray of Newcastle, with very encouraging results. There is a curious contrast in regard to immediate effect between this treatment and the treatment by transplantation. In his patient no change to speak of could be observed for five or six weeks after the treatment was begun, though since then her condition has improved most satisfactorily.

IV. Exhibition of Instrument.

Dr Dawson Turner showed a UNIVERSAL ELECTRIC BATTERY.

V. Original Communications.

1. A THOUSAND CASES OF PULMONARY TUBERCULOSIS, WITH ETIOLOGICAL AND THERAPEUTIC CONSIDERATIONS.

By R. W. Philip, M.A., M.D., F.R.C.P. Ed., Assistant Physician to the Royal Infirmary; Physician to the Victoria Dispensary for Consumption and Diseases of the Chest, Edinburgh; Lecturer on Practice of Physic, Edinburgh School of Medicine.

The considerations which I venture to submit to the Society to-night are based on a careful analysis of the records of one thousand cases of pulmonary phthisis, which have been seen, and, in the great majority of instances, have been treated by me during a lengthened period.

I have thought it well to exclude, for present purposes, a relatively small group of cases where difference of opinion might reasonably be admitted as to the diagnosis of pulmonary tuberculosis—a term which I shall employ in what follows as synonymous with pulmonary phthisis. In every instance the diagnosis rested on a consideration of the patient's symptoms and of the physical signs, a graphic and diagrammatic record of which was kept. In cases of doubt—and, apart from this, in a large proportion of the entire number—the diagnosis was rendered complete by the detection of the tubercle bacillus in the sputum.

In examining for the bacillus, I have generally practised a modification of the carbolic fuchsin and methylene blue method, which I have quoted elsewhere.[1] When the bacillar elements have been scanty, and their detection consequently more difficult, I have fallen back on the precipitation and concentration method, suggested by me to the Society in 1886.[2]

The major portion of the present paper will be occupied with a discussion of assigned or assignable etiological factors, which will be taken up *seriatim*, as may be convenient, and I propose to interweave with these certain general therapeutic considerations, as the occasion may suggest. It is not my intention to enter on details of treatment to-night.

Occupation.—On the general subject of occupation I need say little. The table emphasizes the acknowledged dependence of phthisis on insufficiency of fresh air and exercise, confinement to badly-ventilated and over-heated apartments, the inhalation of dust, and also, though doubtless to a less degree, on exposure to unfavourable and irregular conditions of temperature, frequent child-bearing, prolonged lactation, and the like. A comparison of the first and last columns affords a striking illustration of the

[1] *Pulmonary Tuberculosis.* By R. W. Philip, M.A., M.D. Edinburgh and London: Young J. Pentland: 1891.

[2] *Transactions of Medico-Chirurgical Society of Edinburgh*, vol. v. New Series, 1885–86.

efficiency of occupation as a causal factor. I shall have occasion to refer to the subject in more than one connexion later.

In respect of the group indicated as labourers, it should be noted that a considerable proportion of its members were trained workmen of various classes, whose chest affection and consequent inefficiency, in some instances accelerated by their own irregularities, had reduced to the position of day labourers.

TABLE I.—*Occupations of One Thousand Tubercular Patients.*

Housewives (married),	141	Ironmoulders and Typefounders,	15
Clerks and Warehousemen,	72	Collectors,	14
Labourers,	72	Rubberworkers,	14
Masons,	54	Bakers,	12
Salesmen and Saleswomen,	53	Blacksmiths,	11
Carpenters, Joiners, etc.,	48	Gatekeepers and Messengers,	11
Children, unemployed or at School, up to 18,	42	Hawkers,	11
Printers and Compositors,	39	Painters,	11
Tailors and Hatmakers,	37	Charwomen,	10
Domestic Servants,	35	Coalworkers,	10
Bookbinders and Folders,	31	Waiters, etc.,	10
Dressmakers and Seamstresses,	27	Millworkers,	9
Porters,	22	Fishermen and Sailors,	8
Engineers and Enginemen,	19	Glasscutters and Grinders,	8
Cabmen, Coachmen, and Carters,	19	Plumbers,	8
Brassfinishers,	17	Rivetters and Fitters,	8
Firemen,	6	Dairymen,	2
Tanners,	6	Farmers,	2
Combcutters,	5	Musicians (Wind),	2
Shoemakers,	5	Plasterers,	2
Builders,	4	Slaters,	2
Glaziers and Gilders,	4	Butchers,	1
Police and Watchmen,	4	Clergymen,	1
Students,	4	Engravers,	1
Tin and Wireworkers,	4	Fisherwomen,	1
Bankers,	3	Gardeners,	1
Laundresses,	3	Guards,	1
Merchants,	3	Medical Practitioners,	1
Shunters,	3	Sick Nurses,	1
Teachers,	3	Soldiers,	1
Athletes,	2	Nondescript Adults,	22
Corkcutters,	2		1000

Sex and Condition.—My statistics show a marked preponderance of male cases, in the ratio of 63·7 per cent. male to 36·3 per cent. female. This is different from the ratio which obtains in the United Kingdom in the aggregate, where sex, uncomplicated by the question of age, appears to play a relatively minor part.[1] Dr Ogle, the Registrar-General, gives the mean annual mortality from phthisis (on an average of thirty years) as—Males 2418, and

[1] It is to be noted that those figures have reference to deaths from phthisis, while mine refer to cases admitted for treatment.

females 2428, per million living. But my figures accord remarkably with those of the first Brompton Report, which, taking mortality as the test of liability, gives the ratio as—Males 61 per cent., to females 39 per cent. Dr Pollok's figures are—Males 60·7, females 39·2; and Dr Williams' are—Males 62·5, females 37·5. It is of interest to note that the Registrar-General's returns for the city of London show a similar disproportion between the male and female percentage of deaths from phthisis.

From the marked divergence of the figures (male 63·7, and female 36·3) I think we may fairly admit, without pressing the matter too far, that the presumption is that in Edinburgh males are more frequently the subjects of phthisis than females.

This view is further supported by the returns of the Registrar-General for Edinburgh, which show a marked preponderance of male deaths from phthisis. In 1887, the figures were—male, 263; female, 221. In 1888—male, 267; female, 219. In the returns (1887) from the nine principal towns of Scotland, Edinburgh is the only one which shows this relation between the sexes. In all the others there is a preponderance of female deaths from phthisis. The figures are as follows:—

Edinburgh, . . .	Male	263	Female	221
Glasgow, . . .	„	220	„	272
Dundee, . . .	„	143	„	233
Aberdeen, . . .	„	86	„	103
Greenock, . . .	„	68	„	76
Leith, . . .	„	64	„	66
Paisley, . . .	„	55	„	94
Perth, . . .	„	21	„	26
Kilmarnock, . .	„	26	„	35

Several factors might be cited in explanation of this disparity. The following considerations suggest themselves:—

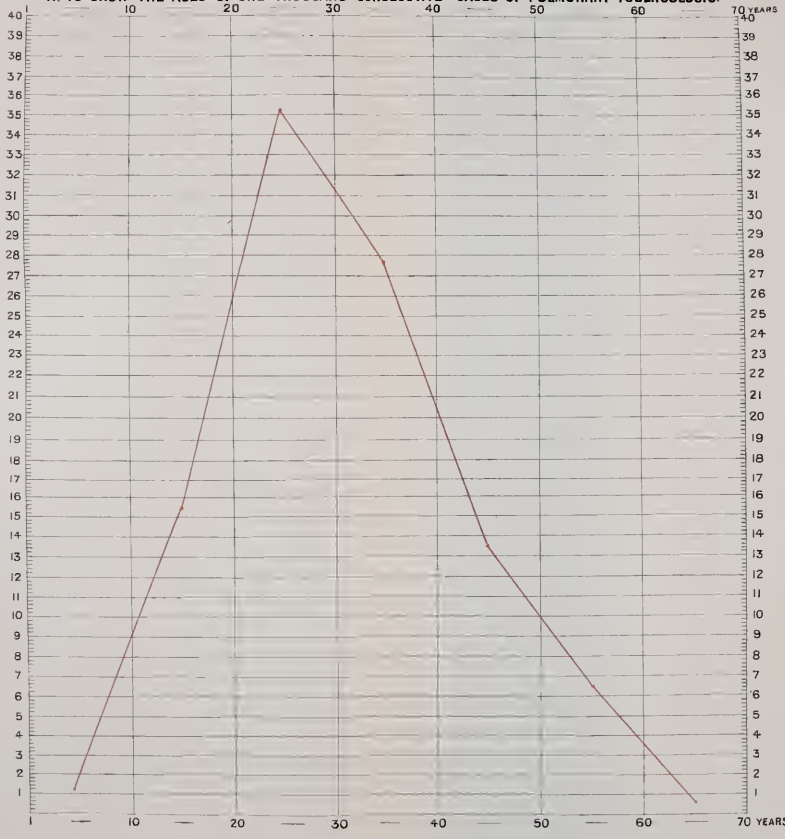
1. *Cæteris paribus*, the phthisical condition is likely to be more quickly noticed in the case of the hard-working members of a community; that is to say, that early depreciation of strength will be sooner observed by such individuals, leading them to seek help. The comparative absence from Edinburgh of large factories and mills, employing great numbers of female hands, will tend to accentuate this element in relation to the male as compared with the female.[1] This might help to explain, also, how it happened, as

[1] It is of interest to note, in this connexion, the relation of the sexes to the industrial occupations in different centres, as these are shown in the Census returns for 1881. Taking the four chief towns of Scotland they are as follows:—

Edinburgh, . . .	Male	42,146	Female	14,034
Glasgow, . . .	„	113,986	„	56,647
Dundee, . . .	„	29,702	„	27,938
Aberdeen, . . .	„	19,775	„	9,014

The proportion of male to female industrial workers is much greater in Edinburgh than in any of the others. A comparison of this table with the phthisis returns just cited will be found instructive.

11. TO SHOW THE AGES OF ONE THOUSAND CONSECUTIVE CASES OF PULMONARY TUBERCULOSIS.



a rule, in the present series of cases, that the progress of the disease was generally less in the male subject, *when first seen*, than in the female. The latter seemed more tolerant, in many instances, of the advance of the disease, because, perhaps, the calls on her strength were less continuous and urgent. The converse would naturally be the case in centres where there was a large demand for female labour of an exhausting kind.

2. There can be no question, I think, that in the female the process, when once established, is, as a rule, more rapid. On this head my observations coincide with those of most other physicians. Other things being equal, I believe the prognosis in respect of duration is in favour of the male by from twelve to twenty-four months. Hence, even should the *mortality* list be more equal as between the sexes, there will tend to be a larger proportion of male chronic patients able to attend as out-patients at a given time.[1] It would appear that the male patient has a greater power of resisting the advance of the disease. I shall have occasion to note later that the term of life during which males are liable to succumb to the disease is a larger one.

With regard to *condition*, my statistics would indicate that there is an almost equal liability to phthisis in the married and unmarried female. In the male they seemed to indicate that the married was more liable than the single (married, 374; single, 263). But the greater part of my cases belonged to the working classes, among whom early marriage tends to be the rule.

While on this topic, I may add that frequent pregnancy and prolonged lactation appeared to me partly responsible for about 6·5 per cent. of all the female cases, or, if we take the cases occurring in married women only, for 18 per cent.

Between the menstrual function and the occurrence of phthisis the only relation I have been able to trace is the evident one of the tendency to suppression of the menses in presence of the disease, and to the aggravation of several of the symptoms of phthisis about the normal period. This was especially seen in the occurrence of hæmoptysis, which it would be hardly correct to term vicarious. A similar aggravation of symptoms has frequently been noticed in presence of constipation, and, in many cases, one of our best remedies for hæmoptysis is a dose of calomel.

Age.—In Chart II. I have reduced to a curve the main features traceable in the combined 1000 cases. In childhood, below 10, there are relatively few cases (1·2 per cent.) During the decade from 10 to 20 there is a comparatively rapid rise to 15·4 per cent., the rise being, however, not equally distributed over the ten years, but appearing especially during the five years from 15 to 20 (from 10 to 15 reaching 3·1 per cent.; 15 to 20, 12·3 per cent.) This is followed during the decade from 20 to 30 by a more

[1] This applies with especial force to such a centre as Edinburgh. See footnote, page 106.

rapid rise (to 35·3 per cent.), the maximum being reached between 25 and 30. Then comes a slight dip for the decade between 30 and 40 (27·6 per cent.), the percentage between 30 and 35 remaining, however, approximately on a level with that between 25 and 30. A more rapid fall takes place between 40 and 50 (to 13·4 per cent.), and similarly between 50 and 60 (to 6·4 per cent.) After 60 the number of cases in my list is relatively small (0·7 per cent.)

The maximum of frequency for the collective number lies between the ages of 25–35.[1]

On analyzing the age ratio of the sexes singly, I have found that the crest of the wave is reached at different points. Taking the male patients apart, I have noted it as being reached between 35 and 40, while in the female it is reached below 30. In other words, the most frequent date for the occurrence of phthisis would seem to be earlier in the female.

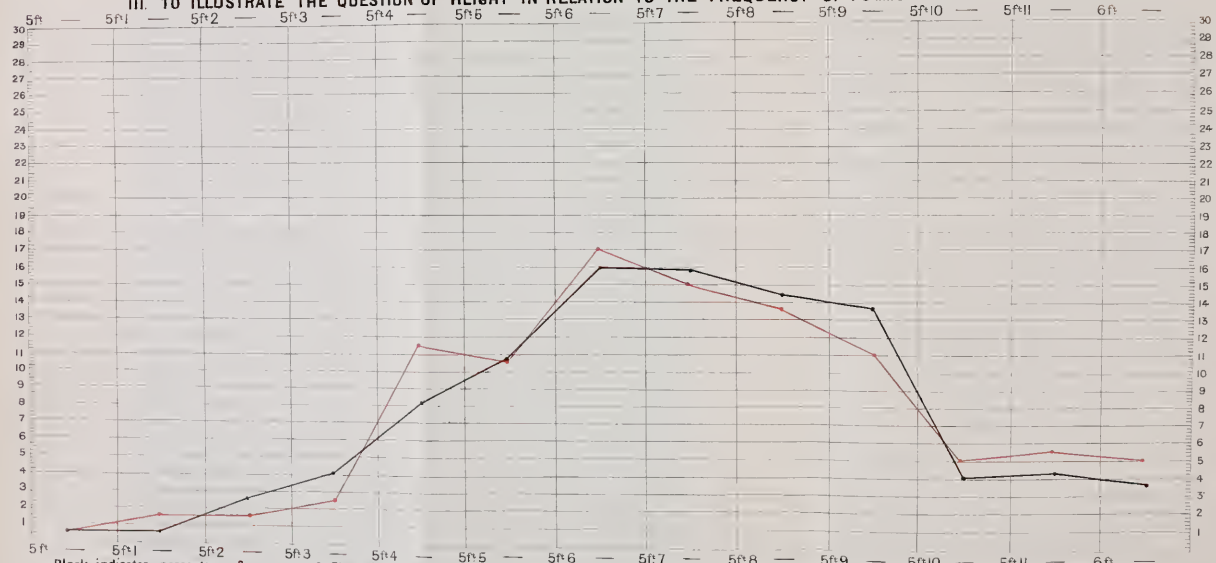
These results accord generally with those of several previous observers, and with the tables of the Registrar-General, where mortality is the test of frequency. They emphasize the fact that phthisis in the male is not so specially a disease of *adolescence* as is sometimes suggested, and that the occurrence of the disease is frequent up to middle life. They would also seem to support the view of the greater chronicity of the process in the male patient, to which I have already made reference. My results will not allow me, however, to go so far as some authors who have indicated by statistics, *e.g.*, that in Germany the maximum of frequency is reached about 60.

While speaking of age, I should add that the general character of the cases has, in my experience, shown a tendency to alter as age advances. After 30 to 35 in the female, and, say, 40 in the male, the type becomes more chronic. But my records include several instances of acute phthisis occurring in patients much further advanced in life.

In judging of the cause of this apparent difference in the age of greatest frequency in the two sexes, one naturally falls back on the difference in time of the occurrence of sexual maturity. Probably this plays *some* part in respect both of the earlier appearance and disappearance of phthisis in the female. But it must be borne in mind, and my cases corroborate this strongly, that in childhood, from 5 years onward, the frequency of phthisis is greater in the female. The determination of the greatest frequency at slightly below 30 in the female, would, so far as my statistics go, seem to be in part explained by reference to the depressing influence of frequent child-bearing and prolonged lactation. As to the earlier diminution in frequency in the female, it is possible that some credit is to be given to the incidence of the climacteric. I am rather inclined, however, to lay stress on the fact that men are, by the necessity of work, exposed during a more prolonged period to the influences of the causes which induce the disease. In other

[1] The chart shows this insufficiently from its arrangement in decades.

III. TO ILLUSTRATE THE QUESTION OF HEIGHT IN RELATION TO THE FREQUENCY OF PULMONARY TUBERCULOSIS.



Black indicates percentage frequency of Phthisical Patients at given heights. Red indicates percentage frequency of General Patients at corresponding heights.

words, the average length of a woman's working life is, from various causes, shorter than that of the male, and when she is subjected to correspondingly prejudicial influences, she succumbs earlier and more rapidly.

Similarly, in speaking of the relation of phthisis to growth and development in the male, it must be remembered that the periods in which phthisis appears more frequently are those also when the strain of continuous hard work is greatest, and the exposure to predisposing conditions most common.

Height.—The question of a possible relation between height and the occurrence of phthisis is worthy of consideration here, in view of the interesting investigations by Dr James, communicated to the Society[1] some years ago. My records have enabled me to compare the height of some 500 *male adults* suffering from well-marked pulmonary tuberculosis, and that of a corresponding number of male adult patients, consecutively presenting themselves for examination and suffering from different affections. I have arranged Chart III. to indicate the percentage number of cases from the two groups at successive heights. The *black* line records the results from the phthisical 500, the *red* line has reference to the other group. A glance at this will indicate that the two curves are approximately similar. It will be noticed that the maximum in both is reached at 5 feet 6 inches.[2] Both continue high till 5 feet 9 inches, after which in both cases there is a sudden fall to 5 feet 10 inches, from which point there is but a slight fall to 6 feet. It will be noticed, however, that the phthisical curve falls further than the other. Dr James's table shows a similarity with my chart in respect of height generally, which corresponds with statistics from other sources with reference to the industrial classes, but it differs from mine in respect of the phthisical group. According to his statistics the summit of the wave of phthisical frequency is reached at 5 feet 8 inches, *i.e.*, 2 inches more than the height of greatest frequency in the male adult, and the phthisical curve remains thenceforward higher than the other till 6 feet is reached.

On these figures Dr James rests the view that tall men are relatively more liable to phthisis than short men, and, while admitting that the number of phthisical cases he has included—about the half number on which the Chart I have shown you was prepared—he elaborates a theory of the relation between tall individuals and a tendency to phthisis with which I cannot get my facts to agree. He goes so far as to say that, "the diminution in the stature of men in the large towns and manufacturing districts of the British Islands must, like all Nature's acts, be

[1] *Transactions of the Medico-Chirurgical Society of Edinburgh*, vol. iv. New Series, 1884–85.

[2] This is of interest as corresponding with the mean height of the artisan class (male adult) as determined by the Anthropometric Committee of the British Association, and by other observers.

regarded as representing, in the circumstances, a salutary process."[1] It is not my purpose to deal critically with other theories just now, but rather to give a summary of results. But I would submit that as the tall man has a greater respiratory capacity and presumably a greater assimilative power, the tall man has something to spare which is not available to the smaller man, and has, consequently, a better chance of withstanding the adverse conditions which tend towards the production of phthisis.

The figures I have presented to you in the Chart would indicate that the relation of phthisis to height is a simple one. The correspondence between the red and blue curves would point to the occurrence of phthisis with practically equal frequency in persons of all heights, the occurrence being determined by other factors rather than by height proper, such as formation of chest, or rather, we may say, disproportion or deformity of chest architecture, and particularly by the conditions of life to which the individual is subjected. I fear that, apart from racial peculiarities and the effects of intermarriage, the smaller stature which has been noted as prevailing in large towns and manufacturing centres is one of the tributes which Nature demands from so-called advancing civilisation, and coupled as it often is with anæmia, indigestion, and other infirmities, is to be regarded as evidence of depreciation rather than of preservation.

Heredity and Contagion.—I have analyzed the records in relation to these subjects and sifted the evidence with care. My statistics show that in 23·3 per cent. a definite family history of tuberculosis could be traced. In some 5 per cent. more there was a shade of dubiety as to the facts or the evidence. In the remainder, *i.e.*, allowing a further small percentage for inaccurate statement, in 70 per cent. the family history was reported as good, so far as tubercular taint was concerned. The proportion then of hereditary predisposition is a small one,—considerably smaller, I think, than is often supposed. I may add that while diversity of statement occurs among the authorities, the consensus of opinion does not increase the percentage much. My figures agree with those of Dr Flint in America and Dr Pollok at home, who give the percentage of heredity in the widest sense as 30 and 24 respectively. The percentage I have quoted includes a small proportion of cases which must be included as examples of probable contagion. I should add that I agree with those who think that the hereditary element is a more evident factor in the female subject of phthisis.

The question of hereditary influence is further complicated if we consider that one-seventh of the whole mortality of Europe is due to tubercular disease. The number of heads of families included in this calculation must be large. If direct heredity had the significance as a determining factor which some have supposed,

[1] *Pulmonary Phthisis.* By Alex. James, M.D. Edinburgh and London, Young J. Pentland, 1888.

we should expect that the number of cases in which it was traceable would be enormously greater. This becomes more evident if we consider how rapidly a man's lineal ancestors multiply as we pass backward in time. Says Blackstone in his *Commentaries*,[1] quoted by Sir James Paget,—"Of lineal ancestors, every man hath two in the first ascending degree, his own parents; he hath four in the second, the parents of his father and the parents of his mother; he hath eight in the third, the parents of his two grandfathers and grandmothers; and by the same rule of progression he hath an hundred and twenty-eight in the seventh, a thousand and twenty-four in the tenth, and at the twentieth degree, or the distance of twenty generations, every man hath above a million of ancestors." Now, correcting this, as suggested by Paget, in respect of possible marriages of blood relations, "we may safely estimate that at 'the distance of twenty generations every man hath many more than half a million of ancestors;' and the estimate of 'above a million,' though inaccurate for the number of ancestors, is necessarily accurate for the number of times of transmission of hereditary properties."

The figures I have cited and the considerations suggested seem to limit considerably the direct operation of hereditary influence in phthisis, as frequently understood. I am far from excluding, however, a certain relationship,—in the sense that parents weakened from different causes are likely to produce a progeny with less power of resistance. I believe we have to deal with the hereditary transmission of less resistant tissues. Sometimes, too, tissues which resist strongly one form of attack yield readily to another. We have only to think of the frequency of rheumatism in certain families, or of their liability to certain forms of infection, as, *e.g.*, the exanthemata. We must bear in mind that a corresponding condition of relative susceptibility or insusceptibility has been frequently recorded of different races of man in the presence of contagion introduced for the first time from without. Similarly one of the difficulties of some departments of bacteriological research lies in the fact of the insusceptibility of certain animals to certain contagions and poisons, and some of the newer departures in the department of therapeutics have rested on the recognition of such principles.

It seems to me we may safely limit the influence of heredity in respect of our present subject to the production in the tissues of a susceptibility to, or at least a diminished power of resisting the tubercle bacillus. The recognition, within recent times, of the specific element has already cleared away some of the difficulties which beset the problem of heredity.

This raises naturally the question of *contagion*. Of the cases under review, 6·7 per cent. afforded definite, and, in my judgment,

[1] Blackstone's *Commentaries*. Ed. Sweet, 1844. Vol. ii., p. 202. See Paget, *The Alcohol Question*. London, Strahan & Co.

not easily controvertible evidence of the existence of contagion. I discussed the "pros" and "cons" carefully in each case, and only admitted such as seemed sufficiently well established.

The series includes the following types:—

1. Where infection seemed to pass from husband to wife. Several cases. In two being double (a second wife falling victim), and in one, the infection apparently passing from the widow to her second husband.

2. Infection from wife to husband. Several cases. In one instance two husbands successively were affected. Infection from wife to husband has, in my experience, been less frequent than the converse.

3. Infection from children to mother. Two cases. One case was especially significant. A mother advanced in years, previously healthy, nursed a phthisical son for several months, became infected, and died after a short illness.

4. Infection from children to father. Three cases. In one instance, a daughter became phthisical at a boarding institution, returned ill to her parents' house, and soon after, the father, previously healthy, became affected. In another case, a son, long ill, infected his father, an old man, who nursed him with much devotion.

5. Infection from step-mother to step-daughter. One case. Step-daughter, previously sturdy, aged 16, nursed step-mother, whose only boy suffered from tubercular disease of bone, through long phthisical illness, and showed signs of phthisis about date of mother's death.

6. Infection from companions, sleeping or living together, not related. Several instances, both male and female.

7. Infection from tubercular virus by external injury. Two cases. One patient had hand poisoned, the wound healed badly, and enlargement of cubital and axillary glands followed. A superficial tubercular affection appeared close to the elbow, warty in form, with cicatricial development. Shortly after a somewhat rapid pulmonary tuberculosis manifested itself.

8. Infection from brother to sister, and conversely. Several instances. This has been more common, in my experience, from brother to sister.

9. Infection from father to children. Several instances. In more than one, the development of the case in the father was slow, in relation to one of the pneumokonioses. Later, the course became more rapid and evidently tubercular. During the latter stage, two daughters (one grown up), previously healthy, became affected with tubercular disease.

10. Infection from mother to children. Several instances.

I have advisedly placed groups 9 and 10 last. As examples of transmission from parent to child, they more readily suggest the influence of heredity. But in recognising a possible hereditary bias, as we have seen, we have only prepared the way for effective

contagion. In the case of parents (especially the mother) and children, the conditions for contagion are particularly favourable. One has to admit—(1), the possible transmission of the bacillus from parent to offspring in utero; (2), such transmission by the milk; (3), infection through kissing and fondling; and (4), from particles of sputum, carelessly disposed in the contracted dwellings of the poor.

The instances of *probable* infection might be considerably increased. But all the cases I have admitted for present purposes have been carefully tested. Along with similar evidence from other sources, they compel me to admit the contagiousness of phthisis, apart from the abundant experimental corroboration which we possess. I am further led to believe that a considerable proportion of cases which have been attributed vaguely to other contributory agencies are thus explicable. The vitiated air of our crowded workrooms, dwellings, and offices is bad, but is chiefly prejudicial because of the presence of the tubercle bacillus. Granted that you have one or more consumptives present in such room, you will have the dispersion of the contagious element perhaps with the breath, certainly sometimes in the involuntary expectoration which attends a cough, and always in the sputum, which is disposed usually anywhere and everywhere, both in workrooms and dwellings.

Hence, while insisting on the erection of large, well ventilated and lighted buildings, it is of the first importance that patients be educated practically regarding the danger which accompanies such indiscriminate expectoration. When possible, spitting must be forbidden, just as smoking is, except into special receptacles containing water, or it may be an antiseptic fluid, the primary object being to prevent the expectoration drying. Further, it seems to me our duty to insist on the exclusion, from crowded workrooms and factories, of patients with pronounced phthisis. I know—and I daresay we all know—of more than one such workshop, where patients struggling with a rapidly advancing phthisis are in more or less regular attendance, to the certain detriment of their own chances, and at infinitive risk, in my judgment, to those who are associated with them. None of us have power to prevent such attendance meanwhile, and the only way to effect the reform is to recommend some system of medical supervision on the part of those in management, or of the city authorities.

Further, in this relation, I am daily confirmed in the conclusion which I ventured to suggest in a Health Lecture[1] on this subject in 1890, that tubercular disease should be included in the list for compulsory returns to our Medical Officers of Health. It seems to me that no great harm could follow this extension of a most valuable method, which, though at first warmly opposed in the case of the other infectious diseases, has been found

[1] *Consumption.* Edinburgh Health Series, 1890.

on all hands to work smoothly, and has proved of immense service in the checking of disease. To illustrate the significance of it in relation to phthisis, I have prepared a map of Edinburgh to indicate the streets and places in which patients coming under my observation with pronounced pulmonary tuberculosis, during some three years, resided. A red dot indicates the residence. I have frequently found that several cases were present in the same stair or house, so that the localisation is only approximate. Some of the older parts of the city show an almost continuous colouring of red. The mere announcement by the medical practitioner of such case need not interfere with the full liberty of both patient and doctor, and would not be followed by any interference in the great majority of cases. But it would make certain that both doctor and patient were alive to the necessities for the adoption of the general principles of disinfection during life and after death, which, meantime, are very largely ignored in connexion with phthisis. I refer more especially to the disinfection of discharges, and of rooms long occupied by dying phthisical subjects. It would probably necessitate—what, apart from this, ought to exist—an asylum or home for dying cases of consumption from among the very poor. This is a question affecting preventive medicine, and is separate from the other question of the desirability of the establishment of a Consumption Hospital, which should exist for the treatment of the disease, more particularly at a stage when there is a reasonable hope of effecting some good. I cannot help thinking that a portion of our City Hospital—failing any other arrangement—ought to be devoted to the reception of such dying cases, whose miserably close and ill-ventilated surroundings are not only unnecessarily hurtful to themselves, but must accentuate tenfold the risk of contagion to the rest of the inmates—perhaps a family of young children. If there be the slightest degree of truth in the contagious view of tuberculosis, such chronic foci of infection ought not to be permitted to smoulder under conditions which are calculated to encourage the fatal propagation.

Passing from this subject of pre-eminent importance, both from the pathological and the therapeutic point of view, I would beg your consideration of some other etiological factors of interest.

First of all, a few words as to the *more frequent seats of pulmonary tuberculosis.* The records confirm the view that in the vast proportion of cases the process is apical from the first. In 2·3 per cent. only had it exclusively or especially a basal commencement. Previous observations have differed much regarding this subject. Thus Cotton and Percy Kidd have separately placed the percentage at 0·2, while Bowditch, Flint, and others have recorded considerably larger proportions,—the last named citing approximately 4 per cent. As to the side more particularly involved, opinions have been also various. My statistics would indicate that

MAP OF EDINBURGH

Showing Distribution of Cases of Pulmonary Tuberculosis received at the Victoria Dispensary for Consumption and Disea

The Edinburgh Geographical Institute

The red dots indicate approximately the residence of the patients. The streets or divisions of streets have been marked with care. For present purposes it has been deemed unnecessary to locali

marked with care. For present purposes it has been deemed unnecessary to localise the cases at particular numbers.

P OF EDINBURGH

at the Victoria Dispensary for Consumption and Diseases of the Chest during Three Years.

there is no very great difference between the two. In 40 per cent. the left side appeared affected alone or in pronouncedly greater degree; the right in 34·3 per cent.; and both seemed approximately equally involved in 25·7 per cent.

Other Assigned or Assignable Causes.—In reviewing the assigned causes and the mode of onset in the thousand cases, the most striking feature has been the apparent absence of any assignable cause in a considerable proportion. Such absence was noted in some 40 per cent. This renders necessary a further reference to the relation of occupation to the production of phthisis. It accentuates the fact that the occurrence of the disease is largely dependent on ignorance of hygienic conditions and laws on the part of our working people, and negligence or powerlessness in their application on the part of those who know better. Edinburgh is a centre where from the relative absence of large manufactories, one might *a priori* have expected that our working classes should have been relatively free from phthisis. The table of occupations shows a high percentage among clerks, warehousemen, salesmen and saleswomen, tailors, and the like. Such patients report that they have been most careful and regular in their life, that there is no family weakness, and so on. But when one inquires into the conditions of their work, the results are less satisfactory. It is carried on in crowded, perhaps artificially lighted apartments, with little or no attempt at ventilation. As to their diet, breakfast is generally bolted or scamped, dinner is not infrequently taken in the establishment, and the day concludes by their returning home too worn out to do much till bedtime, when they fall asleep in a room equally stuffy and contaminated. As a rule, the patient is so ignorant that he fails to realize the relationship between his life and the disease which threatens him, or so apathetic that he cannot grasp the idea that the conditions can be voluntarily improved—in part at least. Thus of the entire series of cases, only 1 per cent. spontaneously attributed his illness to his sedentary life.

Further, as I have already indicated, there is good ground for supposing that one infected person in a given shop or warehouse may be sufficient, in the co-existence of unsatisfactory conditions of ventilation and of dirty habits in the employés, to disturb many calculations. Hence we are bound to insist on the inspection of our shops and warerooms and offices, with a view to prevent overcrowding and insure proper ventilation. But this must be combined with some arrangement for medical inspection—on the part of our employers of labour in the interest of the workers—which would prevent the participation in such indoor occupations by patients suffering from evident pulmonary tuberculosis. This plan would be rendered unnecessary by the adoption of—or would come to be a subsidiary part of—the system of compulsory notification, which sooner or later, in my opinion, must be faced.

However attained, it would in the end be the kindest plan to the individual, guiding him to other departments of work, or to an out-of-door life. It would be a wise policy for the State and community, with a view to the diminution and prevention of the disease. The plan would have the further advantage of conducing to the relatively early detection of cases of phthisis. I know there are serious difficulties, but it seems to me that the time has come for pressing such considerations. I do not for a moment imagine that all is attainable, and I am prepared for further suggestions in improvement or modification of what I have sketched. But I venture to crave the combined and practical aid of the members of this Society in an attempt to obtain for our people the protection which their ignorance and traditional conditions of life prevent their realizing.

Of assigned causes, apart from more general ones, *chill* was cited as the direct agent in 12·8 per cent. This included irregular exposure to extremes of cold and heat in connexion with work, negligent exposure after overheating, or continuance in wet clothes.

In 12·4 per cent. *bronchitis* was assigned as the commencement. Doubtless this was correctly reported in a proportion of the cases. But the relation of bronchitis to phthisis is a complicated one. In patients below par, with feeble respiratory apparatus and depreciated circulatory function, a bronchitis may pave the way to pulmonary phthisis. The bronchitis removes insufficiently: it may hang about as a bronchiolitis, or apparently get fixed as an alveolitis—what has been described elsewhere as an apical catarrh. I need hardly accentuate here the significance of a so-called localized or resident bronchitis, as suggesting—or rather constituting—the initial stage of pulmonary tuberculosis. On the other hand, it is common experience that in the great majority of ordinary cases of bronchitis the event is otherwise. The condition is recovered from completely; or the condition may be recovered from, but a tendency to its recurrence has been established, and the patient has an annual or more frequent attack of so-called winter cough. He may suffer secondarily from circulatory and other disturbance. But he does not, with any great frequency, tend to become phthisical. One recognises, in this connexion, two sufficiently distinct types of bronchitis.

But it is questionable in how far the early diagnosis of bronchitis in some of these cases may have been a correct one, and whether the process, even before the diagnosis of bronchitis was given, was not truly tubercular. From my statistics I have formed the opinion, that the signs of resident bronchitis (localized rhonchi, etc.) are sometimes assigned to bronchitis, when with more complete examination and fuller consideration the diagnosis would have been graver. Those are the cases where examination of the sputum comes to be of special service. There can be little ques-

tion that death certificates are not infrequently filled up with the cause bronchitis where phthisis should have been recorded. I have seen the sudden supervention of pneumo-thorax in two cases with other signs of phthisis, where up to a fortnight before the occurrence, according to the patient's statement, the condition had been spoken of as bronchitis. Lastly, bronchitis was a frequent complication of most of the cases—requiring treatment *per se*—and tended often to aggravate and accelerate the existing condition.

Pneumokoniosis.—In 5·6 per cent. the patient's statement or antecedents permitted a directly traceable connexion between his condition and the inhalation of dust particles. In the large proportion of cases included in my statistics, stone dust was the exciting agent. This is not the place to discuss the pathology of stone-mason's phthisis or other variety of pneumokoniosis. But my experience in Edinburgh leads me to say that in a considerable number of instances, cases which have presumably begun in this non-specific way, and probably presented for some time the features (physical and otherwise) of so-called fibroid phthisis, tail off into ordinary tubercular phthisis—perhaps with more chronic course—as proved by the occurrence of the tubercle bacillus. In such cases I have noted the relative frequency of laryngeal complication. Of symptoms, dyspnœa has seemed to me to assume an unusual prominence in these as compared with other cases of phthisis. Hæmoptysis, while not uncommon, has been relatively less frequent.

Pleurisy.—The onset of the disease was attributed to an attack of pleurisy in 8·2 per cent. In judging of the significance of these figures, all we can say, I believe, is that an attack of pleurisy first drew the patient's attention to the chest. It is much more difficult to predict in how many instances the pleurisy, mostly with some effusion, was the *exciting* cause. I suspect that, as occurs with reference to bronchitis, the early diagnosis is often given to the patient, for one or other reason, in terms of the more evident condition, and the concurrent tuberculosis is omitted. The patient is seen later, and gives a history of preceding pleurisy, but is found to be suffering from pulmonary phthisis. In this respect, the evidence of the dependence of the latter on the former is liable to the same fallacies which exist in judging of the relationship between phthisis and hæmoptysis.

While accentuating the probable tubercular nature of many apparently simple pleurisies, I do not wish to exclude the possibility of pulmonary tuberculosis developing in the wake of a simple pleurisy. I have followed some 1·5 per cent. of the cases under review apparently from the pleurisy stage, and have seen signs of consolidation and softening make their appearance later on in one or other side. There is much *a priori* evidence in favour of a relationship. The respiratory function being impaired, the possibility of withstanding the

tubercle bacillus is considerably reduced. But there is not always sufficient evidence as to the state of the chest prior to the appearance of the "initial" pleurisy; and the frequency with which pleurisy occurs as a complication of phthisis raises grave doubts regarding the sequence of events in all cases. On the other hand, we have all seen instances of pleurisy, apparently idiopathic or traumatic, not accompanied or followed by phthisis. I need hardly remind you of the frequency with which on post-mortem examination the pleural sac is found involved in patients dying from all conditions.

Hæmoptysis.—In 8 per cent. the patients have attributed the commencement of their illness to hæmoptysis. It need hardly be said, however, that it is impossible to accept the patient's statement as having much weight. Often when he is cross-examined it is discovered that other symptoms have really been present. Or in the absence of these, physical examination may show undoubted evidence of pulmonary disease. I fancy we are all agreed that a *phthisis ab hæmoptöe* is a *rara avis*, whose precedents merit an unusual degree of scrutiny. The older view which admitted the occurrence of this rested on a pathology of phthisis which we can no longer accept. The caseation of a simple blood-clot will not now be seriously discussed by pathologists. And the experimental proof that blood (and other fluids) may be effused, accidentally or artificially, into the lung without producing phthisis affords striking commentary. Further, the frequent hæmoptysis which occurs in mitral stenosis, and the relative infrequency of accompanying or complicating phthisis, is important counter evidence. I fear, then, we must admit that the fact of the patient's citation of hæmoptysis as a cause offers little proof. I should add that, in addition to the 8 per cent. we have been discussing, in 18·3 per cent. of the thousand cases which were examined by me, hæmoptysis had occurred once or more frequently after the onset of the initial symptoms, prior to the date of my examination.

Acute Febrile Processes.—(*a.*) *Croupous Pneumonia.*—The present series of cases includes some five (0·5 per cent.) only in which there seemed traceable a close sequence of tuberculosis on croupous pneumonia. I confess I do not feel satisfied that in any one of these we could speak certainly of consequence. I am inclined to think, from my own experience, that the termination of a croupous pneumonia in phthisis, as frequently taught, is an extremely uncommon result. I have seen a larger proportion of already affected chests becoming attacked by croupous pneumonia,—although this, too, has seemed to me less common than, on *a priori* grounds one might anticipate,—and the acute disease has generally run its course naturally. Regarding the supposed cases of phthisis following croupous pneumonia, it would clearly be difficult always to exclude a pre-existing tubercular affection. In fact, it is the more careful examination which convalescence from croupous pneu-

monia in most cases implies which brings to light the other condition. I am of opinion that no close relationship can be proved between them.

(*b.*) *Measles.*—The relationship existing between phthisis and measles is a much closer one. The evidence is clearest and of greatest importance in respect of the young subject. I find that of the cases of phthisis I have recorded as occurring below fifteen years of age, 30·5 per cent. had a history of recent measles. By far the greater proportion of these fell between the ages of twelve and fifteen. There is little doubt that particularly in measles necessary precautions during convalescence are apt to be neglected. Apart from more acute complication, this is too frequently followed by the supervention and continuance of a low catarrhal state of the mucous membranes, which prepares the way for the tubercle bacillus.

(*c.*) *Influenza.*—In 4·4 per cent. the onset of the tubercular disease was distinctly traced to an attack of influenza. I have investigated these cases with jealousy and some degree of scepticism, and have admitted only such as seemed to me to present substantial evidence of the connexion. It is of interest to note that no instance was included in my first 250 cases, and that during last year the number amounts to one-half of the whole. I am left with no possibility but to accept the view which would connect some of the more recent cases of phthisis in considerable degree with the epidemic of influenza through which we have just passed. I am of opinion that ignorance or neglect of the urgently needed precaution during a convalescence, which has been often improperly curtailed, is largely responsible. I should add that, apart from this, a considerable number of tubercular patients have suffered from attacks of influenza as a complication of their already existing disease. This has frequently led to a serious increase of symptoms. On the other hand, a large proportion of tubercular patients have, in my experience, escaped the epidemic altogether. It is an interesting fact in this connexion that, during the height of the influenza epidemic in Edinburgh, an average attendance at the Victoria Dispensary for Consumption and Diseases of the Chest, varying from fifty to sixty patients *per diem*, was not materially affected.

Other Constitutional Conditions.—As to the relation obtaining between other constitutional affections and phthisis, my statistics afford little that is new or significant. In 1 per cent. there was evidence of concurrent *syphilis*, and in something less than 1 per cent. more there was a likely history of syphilitic infection. In 1 per cent. there was a history of acute *rheumatism*, and manifestations of more chronic forms of rheumatism were not infrequent. I have not been able to observe a patient with pronounced tuberculosis affected by a complicating acute rheumatism, so that my experience does not entitle me to confirm or to dispute the alleged antagonism

of their acute phases. Similarly I am not in a position to say anything of *gout* in relation to phthisis. There were no definite manifestations of the former in any of my patients. But gout—more especially so far as dispensary work goes—is relatively less frequent here than in other centres. In 0·7 per cent. there was a history of *ague*, acquired abroad, with, in each instance, the recurrence of attacks apparently after the establishment of the phthisical condition. In none of this necessarily small group of cases did the presence of the one condition seem to exclude or to materially affect the appearance or character of the other. In one case only was *diabetes* a precedent condition. It may be well to mention, in passing, that the patient's sputum contained the tubercle bacillus in large numbers.

Alcoholism was clearly traceable in 1·4 per cent. Considering the large quota of irregular livers which the Dispensary contingent of my patients supplies, I am surprised that the number of tubercular patients was not greater in which such antecedents had to be recorded, either in respect of history or physical condition. I do not wish to exclude the significance of alcoholism as a possible factor, for which a good deal of evidence has been produced in other quarters. But corroboration cannot be obtained from my statistics. In more than one instance I have quoted, moreover, the history of alcoholism was combined with that of privation,—an etiological factor of some importance apart from the other, and a factor whose importance I may here emphasize in relation to the general body of my results, as in the majority of instances, retarding improvement or recovery.

Anæmia and Dyspepsia.—Before passing from the subject of causation, I may be permitted to refer to the frequency with which tubercular disease is masked by prominent symptoms and signs of dyspepsia and anæmia. It is possible to lay undue stress on the statements of patients as to their past history. But I am bound to say that I have met with a seriously large number of cases where, according to the patients' accounts, they had been treated long for dyspepsia or bloodlessness, while examination showed the signs of initial or even pronounced pulmonary phthisis. I feel constrained to emphasize the desirability of exercising the greatest care in concluding that such simpler elements of disease are alone present, and the advisability of renewing the physical examination, from time to time, with the view of obtaining corroborative assurance.

Complications.—Sufficient reference has been made to the close relationship existing between pulmonary tuberculosis and pleurisy, bronchitis, and hæmoptysis (*v. supra*). It is hardly necessary to do more at this point than to note that these were frequently occurring elements of complication in the cases now under review. Of the common complication of phthisis with emphysema and hepatic enlargements of various kinds I do not propose to

speak, but prefer to limit my remarks under this head to certain laryngeal, cardiac, intestinal, glandular, and integumentary complications.

Larynx. — The larynx showed undoubted complication in 10·5 per cent.; and there was a suspicion of affection in some 6 per cent. more. The commonest seats of change were the aryteno-epiglottidean folds and the inter-arytenoid commissure. In a large proportion of cases this assumed the form of a pale boggy infiltration, with or without ulceration. The characteristic pear-shaped swelling of the former region was observed in many instances, apart from loss of substance. In the case of the posterior wall the ulceration was more frequent. The edges of the ulcer were ragged, the surrounding parts were not, as a rule, much reddened, and the gray-looking floor was frequently coated with muco-purulent element. Infiltration of the false cords (with relatively seldom ulceration) and ulceration of the true were not uncommonly noted. Ulceration of the epiglottis, especially of the under aspect, with œdematous swelling of the whole structure, accompanied by grave dyspnœa and dysphagia, was noted in a much smaller group of cases. Such a change implies generally an advanced process, and points to the approaching end. Two patients presented themselves for the first time with such a lesion, and in both instances death followed within a fortnight. In one or two instances the course was more protracted, but the termination was similar. In addition to the more conspicuous changes which have been described, I should add that the larynx was observed to be unduly pale in more than 50 per cent. of all the cases under review,—a pallor which it shared with the adjacent mucous membrane of the palate and palatal arches.

Heart. — Special valvular affection was recorded in twenty instances, or 2 per cent. In seven cases, this took the form of mitral incompetence, in three cases, of mitral stenosis, and in six, of combined mitral stenosis and regurgitation. In the remaining four instances, there seemed evidence of pulmonary stenosis. Concurrent aortic disease was not reported in any instance in the present series of cases. These facts illustrate the comparative infrequency of concurrent phthisis and cardiac disease. But they indicate that the latter does not preclude the former condition. My experience has further led me to conclude that cases of phthisis with concomitant mitral disease tend to be of relatively slow progress. In three of the instances I have quoted, where mitral stenosis and regurgitation were present along with well-marked tubercular affection of the lungs, with abundant tubercle bacilli, the course was unusually protracted, and I was enabled to follow them closely for several years.

Glandular, Intestinal, and other Complications.—It has surprised me to note the comparatively infrequent conjunction of evident glandular affections with phthisis. Of the thousand patients

under review only some twelve, or 1·2 per cent., presented such a condition. In something under 1 per cent. was evidence of tubercular bone or joint affections reported, and a similar number manifested skin affections of tubercular nature, including lupus. A slightly greater number of patients, hardly over 1 per cent., suffered from fistula in ano. In every case those were of the male sex. Diarrhœa was a frequent complication. Very intractable diarrhœa, more evidently tubercular, occurred in from 2 to 3 per cent.

Results.—On the present occasion I must limit myself to submitting a summary of the results of treatment. In preparing this I have excluded from consideration all cases which were followed for too short a period to justify a statement of results. In 469 instances the progress was noted at intervals continuously through six months, and in the greater proportion for much longer. What follows has reference to those patients only. In judging of results, it is to be further remembered that most of the patients belonged to the poorer out-patient class, in whom the possibility of successful treatment is seriously prejudiced by pecuniary and hygienic difficulties.

So far as they go, the results have been encouraging. For the sake of convenience I have arranged the cases in four groups, in terms of the results:—(I.) Good, (II.) Improved, (III.) Indifferent, (IV.) Bad.

(I.) *Good*, including those cases only, where continuous betterment in symptoms, physical signs and general condition, was observed during a period of not less than six months. In many instances the cases were followed for one or more years. The chief test of general improvement was the weight, and only such cases were admitted into this first group as maintained an increase of weight of more than 5 lbs. In a considerable number this amounted to over 14 lbs.; in some to twice or thrice that amount. The change in physical signs was tested by reference to the chart of the patient's condition, which was made when first seen. Regular notes were kept of the changes from time to time, as also of the symptoms. This group includes 117, or 24·94 per cent.

(II.) *Improved*, including cases, where improvement was definitely recorded, but to a less degree; where, for example, the increased weight was less constantly maintained, or to a less degree, or where the amelioration in symptoms or physical signs was less regular. No case was admitted into this group where the physical signs did not show material improvement. This group includes 163, or 34·75 per cent.

(III.) *Indifferent*, including cases, where the patients—observed during a prolonged period—fluctuated from better to worse, and

from worse to better, but did not make steady progress, and on the whole tended downwards. This group includes 113, or 24·1 per cent.

(IV.) *Bad,* including cases, where there was a progressive loss of weight, and more or less rapid advance of the signs of the disease, with sometimes the supervention of urgent symptoms, leading to death. This group includes 76, or 16·2 per cent.

The President thanked Dr Philip for his paper, which was the outcome of an immense amount of careful work, and would doubtless receive, as it deserved, much attention.

Dr James Ritchie asked whether, among so large a number of cases, Dr Philip had found histories of early and excessive menstruation? One of the charts exhibited showed the largest percentage of cases of phthisis between the ages of 25 and 35, but the number of people living at those ages was greater than for the more advanced periods of life,—can Dr Philip give the percentage in relation to the number living at the several ages? In another chart it was seen that the largest number of cases occurred in persons of heights 5 ft. 6 to 5 ft. 8 in., but these are the most common heights in this country,—can Dr Philip state what is the ratio to the numbers alive at each height? In the experience of life assurance companies pleurisy is a very common incident in the histories of those dying of consumption. Pleurisies recovered from are supposed to be frequently tubercular, and that the poison remains latent, but at a later period, under suitable circumstances, it is supposed to become active and to develop afresh,—had Dr Philip many cases with a previous history of pleurisy? The speaker was not sure if he exactly understood Dr Philip's view of the relation of bronchitis to phthisis. He had referred to errors of diagnosis, but he supposed that Dr Philip regarded bronchial catarrh as a predisposing cause by producing a favourable nidus for the bacillus, also that measles acted in a similar way, having, in addition to the bronchial catarrh, the powerfully debilitating influence of the disease. As to hæmoptysis, the speaker's experience was at variance with the views which they had just heard. He believed that hæmoptysis occasionally had a causal relation, that the inspired blood lighted up a pneumonia which became the seat of tubercular infection. Had Dr Philip, in this large number of cases, found that a rheumatic constitution modified the course of the disease in a favourable manner?

Dr R. F. Leith asked if Dr Philip had seen many cases of pneumokoniosis set up by flour, and cited a case seen in the post-mortem room.

Dr R. W. Philip thanked the members of the Society for their kind reception of his paper. Referring to what had been said on the subject of menstruation, he had nothing to add. The curves

which he had prepared to illustrate questions of age and height were especially of comparative and local value. The former indicated that the age of greatest frequency in the Edinburgh district, so far as could be gathered from the statistics of patients still in life, with pronounced tubercular lesions, corresponded largely with the results of the observations made in the United Kingdom generally on the basis of mortality per so many persons living. The curve of height had been prepared, as indicated in the paper, for purposes of comparison with a table communicated by Dr James to the Society some years ago. As to pleurisy, he had endeavoured to emphasize the suspicious character of a large proportion of pleurisies, not as leading to tubercular affection of the chest, but as being the first definite indication of the tubercular condition. Similarly, while bronchitis might lead in the way he had indicated to the establishment of tuberculosis, he wished to accentuate the fact that frequently cases were diagnosed as being simple bronchitis, while the localisation of the rhonchi and the alteration of the breathing in a limited area gave sufficient proof that the process was a more serious one. He had not unfrequently seen such cases where a diagnosis of bronchitis alone had been given, and examination showed that the sputum contained the tubercle bacillus in abundance. He did not think that a hæmoptysis, pure and simple, was probably ever the *cause* of the establishment of the tubercular condition. There occurred occasionally an antagonism between rheumatism and phthisis. Such cases had been recorded. He had been trying for long to get hold of good illustrations of the co-existence of the conditions. Apart from cases of chronic rheumatism, or patients with a rheumatic history, he had not been able to make observations. In these cases there had been no appreciable alteration from the usual course or type of phthisis.

2. *Dr A. Lockhart Gillespie* read his paper on NOTES ON TOXIC EFFECTS OF EXALGIN, of which the following is an abstract:— A young man of 23, suffering from severe toothache, had taken 36 grains of exalgin between 2 and 11 P.M. Shortly after 11 he relapsed into unconsciousness. When seen, his temperature was found to be normal, his pulse 79, regular, of fair tension, but all his muscles paretic. With severe pain in his head, convulsions occurred every few minutes, and on tapping the scalp or pricking the arm or leg with a pin, fresh spasms were initiated. All his reflexes were abolished, and his pupils, widely dilated, reacted sluggishly. As time went on the breathing became of a distinctly cerebral type, the pulse now and then intermitting during a paroxysm. About half an hour after he was first seen $\frac{1}{4}$ grain of morphia was injected, and he swallowed, though with difficulty, two pills, the only purgative handy. Cloths, soaked in cold water, were applied to his head. As the pupils con-

tracted under the influence of the morphia the fits became less severe, but the effect of the drug did not commence until ninety minutes after injection. All the following day the patient, who remembered nothing of the previous night, was unable to use his muscles, while his arms and legs were absolutely anæsthetic. His bowels had moved freely. He had a slight return of the symptoms next night, but 15 minims of laudanum, repeated every four hours, soothed his irritated medulla, and in the course of the following day he became considerably better. Dr Gillespie was inclined to believe at first that the symptoms might be due to a cerebro-spinal meningitis of influenzal origin, as several cases of this disease had lately been seen in Edinburgh. The absence of high temperature and the quiet pulse negatived this supposition. It was then first found out that he had taken so much methyl-acetanilide, a drug the maximum dose of which Bartholow gives as 12 grains *per diem*. The usual treatment recommended for such cases is free stimulation. In this instance it appeared to Dr Gillespie throughout that the reverse was required; and although it might be heroic to inject morphia into an already unconscious man, the end justified the means. Administration of chloroform or ether was out of the question, when the state of the breathing was considered. Other cases have been recorded in the course of the last two years, the symptoms usually comprising delirium, unconsciousness, and convulsions. In conclusion, exalgin was not a safe drug for patients to take without medical advice. It should never be given as an antipyretic, and as an analgesic only in doses of 1 to 3 grains once or twice in the twenty-four hours.

The President thanked Dr Gillespie for his careful description of an interesting and instructive case.

Meeting VII.—March 22, 1892.

Mr Joseph Bell, *President, in the Chair.*

I. Exhibition of Patients.

1. *Dr Michael Dewar* showed a patient with UNUSUAL DENTITION. J. S., aged 35, who, though not altogether an edentate, yet was almost one. In his own words, "he has all the teeth ever he had." He had no temporary teeth whatever. When four years old, he was taken by his mother to the doctor, who lanced his gums, probably as a "placebo." The permanent teeth which had developed were ten in number, viz., two bicuspids and first molar on each side in the upper jaw, and second bicuspid and first molar on each side in the lower jaw. The question naturally arose how in the absence of the milk teeth there could be development of the bicuspids, seeing that the sacs and follicles of the

anterior ten permanent teeth were formed almost simultaneously in the early fœtal months, and in conjunction with and behind the milk germs. The permanent molars, as we know, are developed independently of the temporary teeth. As regards heredity, the evidence was negative. His parents, brothers, and sisters, had all their natural complement of teeth, and, as far as could be learned, the grandparents also. These cases are not very common,—at least there are not many recorded. Mr Cooper of Edinburgh supplied many years ago a full dental set to a young lady who had no teeth whatever. The speaker was informed a few days ago that an Inverness dentist had a whole Highland family under his care who were edentates, but as the history could not be got, the accuracy of the statement could not be vouched for.

Mr Joseph Bell mentioned a case which in some respects resembled that of Dr Dewar's.

2. *Mr Caird* showed—1. A boy, aged eleven, who had been violently struck on the head by a swing boat, which caused a depressed compound comminuted fracture of the right frontal region. Brain matter was scattered over the head, and two fragments of skull were removed from the frontal lobes. After careful purification of the wound, the dura was sewn and the fragments replanted. Sepsis, however, followed; one portion of bone was removed within a week, and the second portion was cast off in a couple of months. The hernia cerebri which had formed rapidly diminished after the bone came away, and the boy, lively and intelligent, exhibited no ill effect from his accident. There was a large pulsating scar. It was proposed that he should wear a protecting shield, but at some future date an attempt might be made to cover in the gap by an osteo-plastic operation. 2. A patient on whom Chopart's amputation had been performed after a crushing accident. The wound healed by the second intention, but the result was excellent, there being absolutely no pointing of the foot, and this although the tendo-Achilles was not divided.

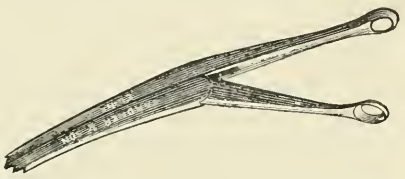
II. Exhibition of Specimens.

Dr Michael Dewar showed—1. A large-sized HOUSEHOLD PIN, which was swallowed by an infant eighteen months old, and passed per rectum, point first, in 61 hours. 2. A small DENTAL PLATE, which was swallowed by a lady while asleep, and passed per rectum in 55 hours. The treatment consisted in the exhibition of soft, bulky, farinaceous and vegetable food, and no medicine.

III. Exhibition of Instruments.

1. *Dr P. M'Bride* showed a NEW FORM OF LARYNGEAL FORCEPS, and said,—It is with some reluctance that I venture to add another

to the already multitudinous varieties of laryngeal forceps, and I only do so because the instrument about to be described seems to me to present certain advantages of a decidedly practical kind. Neither in the curve of the instrument nor in the joint (of the kind known to surgeons as the crocodile) is there anything of novelty, and I shall therefore confine my description merely to the extremities of the blades. These are circular and fenestrated; each consists of a ring of steel, and the inner margin of this has on either side a cutting edge. As will be seen from the accompanying drawing, the outer margins are bevelled in such a way that they project on every side, and thus prevent anything excepting a projecting growth or body being grasped between the inner or cutting edges. In using Mackenzie or Gottstein's forceps it is always possible to pinch up a portion of healthy mucosa, but with the instrument described this would only be possible were it applied to the ary-epiglottic fold—an accident unlikely to happen in the manipulations of one endowed with even rudimentary laryngoscopic dexterity.



In removing a portion of a malignant tumour for microscopic examination I have found these forceps most useful, as by their aid a piece can be cut, or rather punched out from a projecting surface without the addition of the tearing necessary when Mackenzie's instrument is employed. I have also by their means succeeded in removing a portion of a subglottic growth; but in this case recourse was afterwards had to Voltolini's sponge method, by which the main bulk of the neoplasm was detached. It has appeared to me that an instrument made on a similar principle might be used with advantage to extract foreign bodies from such passages as the œsophagus, trachea, urethra, etc.

2. *Dr R. M'Kenzie Johnston* showed an ELECTRICAL AID TO HEARING.

IV. Original Communications.

1. *Dr Alexander Miles* read a paper on THE MECHANISM OF CEREBRAL CONCUSSION. After briefly reviewing the older theories of concussion, he went on to consider the view that the symptoms were due to a reflex vascular disturbance whereby an anæmia of the brain was produced, which led to all the phenomena of the condition. He believed that the cerebro-spinal fluid, which surrounds the brain, fills the ventricles, and surrounds the bloodvessels and nerve-cells, was thrown into a violent state of agitation when the cranium received a blow; and that this fluid-wave impinging on the parts in the region of the bulb, produced a reflex spasm of the

cerebral bloodvessels, whereby the brain was rendered anæmic. He described certain experiments which appeared to prove that the hæmorrhages, gross and microscopic, which invariably accompanied cerebral concussion, were produced as a result of this cerebro-spinal fluid-wave. On *rapidly* aspirating the cerebro-spinal fluid from the skull of a rabbit, he found that hæmorrhages of varying sizes took place all through the brain substance, into the sub-arachnoid space, and into the ventricles, in no way differing from those resulting from a severe blow. When the fluid was *slowly* aspirated, and the bloodvessels thus given time to accommodate themselves to the loss of support, no hæmorrhages were found; and when a severe blow was dealt after the fluid had been withdrawn, the brain and its membranes exhibited no hæmorrhages or lacerations, thus evidently showing that the cerebro-spinal fluid was the essential factor in the production of these lesions. To demonstrate that the wave set up in the fluid was the cause of the hæmorrhages, one eye of a rabbit was enucleated and a fatal blow dealt on the occiput. In the sheath of the normal eye there was found a large blood-clot, while on the other side, where the cerebro-spinal fluid could freely escape from the severed nerve-sheath, no such clot was present. Dr Miles's conclusions were briefly—(1.) That the group of phenomena commonly known as concussion of the brain was the result of a temporary anæmia of that organ; (2.) That this anæmia was the reflex result of stimulation of the restiform bodies and other important centres in the region of the bulb; (3.) That these parts are stimulated by the wave of cerebro-spinal fluid which rushes through the aqueduct of Sylvius, the foramen of Magendie, and from the sub-arachnoid space of the brain to that of the cord, when a severe blow was dealt over the skull; (4.) That the hæmorrhages found throughout the brain substance and on its surface were to be ascribed to the recession of the cerebro-spinal fluid, which naturally supports the bloodvessels of the cerebrum; (5.) That the cerebro-spinal fluid-wave of necessity affects the ultimate nerve-cells which were normally suspended in the fluid; (6.) That the hæmorrhages, while they doubtless give rise to symptoms, were not the proximate cause of those of concussion as usually met with. They are rather "to be looked upon as an index of the force of the blow than as the cause of the resulting phenomena."

The President expressed his admiration of the scientific method and great industry which was shown in the paper.

Prof. Struthers had listened to Dr Miles's paper with very great interest and instruction. He could not but admire the completeness with which Dr Miles had followed out the water passages of the brain, and appreciated their importance. It was quite interesting to him to find parts which he remembered as having been regarded as only a subject for the curiosity of anatomists now come to be regarded as of so much importance in pathology and surgery.

Mr A. G. Miller had listened with much pleasure to Dr Miles's paper, but had no criticisms to offer.

Dr Clouston said that in his opinion Dr Miles laid too much stress on the effects of the brain anæmia, and too little on the direct effect of sudden impact and violence on nerve action, whether conducting through the white fibres or the energizing of the gray matter. We knew that such impact had a directly paralyzing effect on nerve actions, and that the anæmia and the congestion and the ruptures were due to a previous paralysis of the vaso-motor centres which exist all over the brain. In fact, the circulatory effects were secondary and not primary. He urged Dr Miles to repeat his experiments, and, further, to definitize them by using regulated and measured impacts by means of delicate instruments. He pointed out that the intracranial contents were compound, consisting of fluid and solid contents, and that both must be taken into account in explaining the effects of impacts. He pointed out that the immediate and instantaneous mental effects of impact could only be explained by direct action on the nervous tissue. He referred to the importance of Dr Miles's experiment of aspirating the cerebro-spinal fluid, and concluded by congratulating Dr Miles on the industry and ingenuity he had displayed in this important investigation.

Dr P. M'Bride begged to thank Dr Miles for his most interesting paper, and asked a question as to the explanation given by Dr Miles of the escape of cerebro-spinal fluid from the ears without fracture.

Dr Miles, in reply to Dr Clouston, stated that the instrument used to inflict the blows was a Kaffir-stick, so that the point of contact between the skull and the knob was a very limited one, and therefore the force of the blow was concentrated on the point of impact. He pointed out that Duret's experiments were unsatisfactory, in so far as they consisted in forcibly injecting coagulable materials into the cavity of the skull, a method which was in no way comparable to the method in which head injuries are sustained. In other cases Duret dealt *numerous* blows to the skull. He pointed out that Roy and Sherrington's experiments were fallacious, as they provided for a free escape of the cerebro-spinal fluid during the experiment. He emphasized the point that the nerve-cells were acted upon through the medium of the cerebro-spinal fluid in which they floated, and that the cerebral circulation was reflexly disturbed by the wave of fluid stimulating the parts in the region of the bulb. In reply to Dr M'Bride, he stated that his reference to the escape of cerebro-spinal fluid from the ear while no fracture of the base existed was incidental, and had no direct bearing on his argument, but that the mechanism under discussion threw some light on those clinical cases which have been recorded by Bryant, Hutchinson, Battle, Le Gros, Clerk, and others, in

which welling from the ears was a prominent symptom in the absence of basal fracture.

2. THE DIETETIC TREATMENT OF OBESITY.

By W. Towers-Smith, M.R.C.S. Eng.

It may perhaps be assumed that at all times in the world's history individual men have been troubled with the production of excessive fat, and it is consequently probable that even from the beginning more or less intelligent efforts have been made to deal with it. It would be interesting if it were possible to trace the disorder and its therapeutics to this primitive state, and I am tempted to suppose that the research would be fruitful, not merely of all decisive results, but in a high degree also by throwing a bright light on many interesting problems connected with nutrition and metabolism. In his savage state man can have had little tendency to grow fat. His constitution, unimpaired by vicious habits, must have sufficed for the supply only of his natural healthy wants, while the circumstances of his position precluded at once the possibility of self-indulgence, and enforced a life-long system of bodily asceticism. In the earlier civilisations of the East and of Eastern Europe we can describe a remnant of the primitive necessity in the self-imposed restraint of more opulent peoples. The athletic exercises of the Homeric heroes are the corollary of their simple if generous notions of the occasional feast. The abstemiousness of the Spartans was an institution jealously guarded amongst that brave people as the essential condition of their national supremacy. In the far East, where the human product of gentle conditions both of wealth and of climate were first threatened with the insidious corruption of luxury, wise and despotic rulers, no doubt seconded by the general feeling of the subject races, took infinite pains to secure the efficiency of manhood. We read in the *Cyclopædia* of Xenophon of the almost incredible hardships which were willingly submitted to by the Persian youth. They courted fatigue as the source and origin of honour which was to be reaped only in the battlefield, and so far did they carry their notions of training, that the suggestion of any emunctory other than the skin conveyed with it the idea of reproach, and it was a disgrace for a young man even to blow his nose. Such excessive precautions, however admirable as an institution, implies a commencing departure from the earlier condition in which the wants of man's body were so nicely balanced with its usual demands. And perhaps I may be permitted the speculation, that in the spiritualized population of Athens, amongst which the arts and letters and all the higher refinements of which man's nature was capable flourished in a degree which has never since been attained, we are permitted to see the true apotheosis of the natural man.

The state in which the transition to another, whether more or less perfect being, had not yet begun,—the state in which, while the simpler habits of the animal life were retained, the mind was untrammelled by compulsion of the body, and was free for loftier flights in its proper sphere. We are almost certain that obesity was hardly known in ancient Athens. The history of the later civilisations is a production of that of Persia. In the period of vital energy, which was also the period of ascendency, there was a constant struggle against luxury: in decline, degeneracy, and an abject submission to the demands of sense. In either state—whether in that of youth or of decay—we have evidence that, unlike their primitive ancestors, the men of the Western civilisations were subject to a craving of which the immediate effects must be reckoned unhealthy. But this craving, if it can be shown to be progressive, must surely be the expression of an equally progressive physiological change. Its outcome is a tendency, more or less pronounced in different races, to produce excessive fat, to acquire "flesh and all the ills that flesh is heir to." And if these ills must be regarded as intimately connected with a subtle but progressive change within the body, and if that change, as we cannot doubt, is one of the most obtrusive elements in the development both of the individual and of the race, it may fairly be claimed that the management or treatment of obesity is amongst the most imperative duties of the physician. I might dwell upon this position and enforce it from other points of view,—points of view from which the case appears even more urgent, but on that account I am the less tempted to enlarge upon them.

Consider one of those extreme cases with which every practitioner is only too familiar—a case in which age or the debility of chronic illness is thrown in the scale against the sufferer—when, with his organs diseased and impaired vitality, he has need of every effort of which his system is capable to meet some slight, perhaps unexpected, strain. It is then that we think with apprehension of the feeble and overloaded heart, the hampered respiration, the vast extent of useless and cumbersome tissue, which like a parasite draws from the labouring organs the nourishment that they sorely need. That is a sign that he who runs may read. The tendency to cause death is the touchstone of the medical judgment. Here it is apparent, and all the more so because little can be done to meet it. The time for that has gone by; it was a time when the patient was esteemed to be in good health only, because comparative youth and vigour enabled him to draw upon and to waste his surplus vitality, feeding the parasite while he fed himself,—suffering, it is true, but heedless of the future struggle when every ounce of fat would be a weapon against him. Then should have been foreseen the fatty heart, the loaded liver, the restricted lung surface, the kidneys prone to disease, and the inevitable

day when the balance will kick the beam. The tendency to death is made obvious by experience. It is the duty of the physician to forecast events, and I submit that he is as little justified in neglecting to try conclusions with obesity while there is yet time, as he would be if he withheld the stimulant or depletory measures which he tries ineffectually to resist the fatal issue. Surely it is illogical and something worse to overlook at an early stage, where it may be checked, a state of things which becomes at once dangerous and intractable later on. But there is a difference in our notion of disease corresponding with the point of view from which we look at it. The scientific conception is one thing, the practical or working conception another. The latter is that which we first form for ourselves and then infect the public with, and public opinion again reacts upon us. For practical purposes disease includes just those morbid states which the doctor professes to cure or benefit,—where, in fact, he is allowed to try his skill, and where he thinks he can do some good. Now it is in this working category of diseases that I ask for a place for obesity; and to that end I hope, by publishing the results of treatment in my hands, to establish for it the requisite qualification of being amenable to therapeutic measures.

It is true I am not the first who has sought to show this. We live in a country where the conditions of existence incline men to grow fat, and perhaps it may be asserted that the proclivity is inherent in the Teutonic race. It happens, in consequence, that the problem has thrust itself forward automatically; and that, in addition to the small number of thoughtful physicians who have appreciated the need of combatting at the outset a tendency so directly fatal in the end, there have been amongst the vulgar very many sufferers whose present inconvenience has stimulated them to seek means of relief. Many systems have been vaunted and have been in vogue at different times, but none of them have held the field for long.

It will serve some purposes to inquire into the causes of this. The first and the most obvious is the difficulty with which the observer is necessarily met at the outset. Every attempt at a cure involves some degree of privation and no small share of perseverance and resolution. It is too much to say that obesity is always associated with self-indulgence, or even with indolence, but in a conspicuous proportion of cases this is undoubtedly so; and it follows that while self-restraint and temperance are in every case called for, the subjects are mostly those who are least able to bring these qualities to their support. A suitable dietetic treatment must therefore be as easy and agreeable as possible, calling for the minimum of sacrifice, and extending over the shortest possible time. These conditions, so far as I know, have not been realized. The system which bears the name of Banting was said to require a very long time to produce its effect, and it imposed intolerable hardship.

Above all, its restraints were permanent. Banting's principle was, in the main, correct, but his knowledge of diet and its capabilities was defective, and his measures half-measures. Salisbury's plan, at present much in use, is open to the same objections, and it requires, further, a repulsive and ostentatious observance of details, a nauseating and monotonous diet, and a disregard of the claims of the palate which cannot fail to disgust. Here, again, the restraint is never relaxed—a point, in my opinion, of the first moment.

I will allude to the method employed in Germany by Schweninger, Prince Bismarck's attendant, because it may be classed with the others as involving unnecessary hardship; but I will go further and say that, if my views are correct, the abstention from fluid which is its main resource is positively harmful and dangerous:—

"With the exception of Towers-Smith, all make a great point of restricting the quantity of fluid drunk. This is probably a mistake, and all the reasons which have been urged in its favour reveal an erroneous knowledge of physiology. In reality there can, I think, be but little doubt that it is an advantage to take large quantities of fluid, for, as we wish to accelerate destructive metabolism, it seems but natural that we should, as quickly as possible, try to wash out all the products of destruction. By permitting, or better still, by enforcing the drinking of a great deal of fluid we prevent the thirst of which patients so often complain. The diet for the reduction of corpulence should not contain any alcohol, for it can do no good; and many forms of it, as sweet wines and beer, lead to a rapid accumulation of fat. If the patient insist on taking it, a little pure spirit and water is the best he can drink."[1]

I shall endeavour to show that the dietetic treatment which I employ is free from the gravest of these objections, is open but in a slight degree to any of them, and that the results obtained by it are very much better than any which have been claimed for the others.

A diet administered for whatever purpose should be effective of that purpose; it should also be agreeable to the palate. Of this I shall speak later. If it involves restriction, this should be as easy as the object in view will permit, and the period of privation should be the shortest possible. Interminable privation is an absolute bar. Another requisite for a satisfactory diet is that it should be—and should be known to be—wholesome and safe. There must be no fear lest in combatting a remote danger we induce another which is worse or more immediate. If such a danger exists it is a disqualification. If there is any doubt about it the case is nearly as bad, since medical authority will very

[1] *A Text-Book of General Therapeutics* (Dr Hale White, of Guy's Hospital) p. 115.

properly decline to give its sanction to a mode of treatment which is questionable in this respect.

I propose to address myself to this subject first.

The object of treatment, of whatever kind, must be to prevent the formation of fat within the system, and by this and other means to promote the absorption of that which is already there. The obvious means of doing this is to withhold those kinds of food which are especially fat-making. As to what those foods are there are certain data furnished by physiology,—data, however, which must not be regarded as absolutely and necessarily reliable, but which need to be controlled by experience. The truth of this statement will appear later. In addition to withholding fat-forming foods it is necessary to administer others capable of serving the purposes of nutrition, and not in themselves dangerous. Upon this point also physiology has spoken, but I think prematurely and without due regard to unquestionable facts. Thus, for instance, it is commonly laid down in works on diet that the maintenance of the healthy functions requires four parts carbonaceous food (fat and carbohydrates) to one of nitrogenous (Parkes, Lyon Playfair, Pavy, J. K. Chambers). My experience is totally at variance with this. I have personally, or through other medical men, administered a diet of purely nitrogenous food to a thousand and ninety-two persons for a considerable period in each case. Amongst those cases were representatives of every class of life ; and it has been my invariable experience, as it is to those who have worked with me, that concurrently with the rapid loss of weight there was improved nutrition, health, and vigour, with a general sense of well-being to which the subjects had in many instances long been strangers. If my testimony is not enough, I will depose to that of the distinguished physiologist mentioned above, the authority of Dr Broadbent, who, in his recent Lumleian lecture (*vide Lancet*, March 21st, 1891), says that in his experience a nitrogenous diet, largely diluted, is perfectly safe ; and if further evidence is needed, it can be found in the fact that whole races have subsisted on animal food alone, and not only subsisted, but maintained a high standard of bodily vigour (*vide Lancet*, Dr Herschell, February 10th, 1890). The Terra del Fuegans recently exhibited at Westminster are good specimens of human beings fed on a diet entirely confined to lean horseflesh and water. The question arises, How are these facts to be reconciled with the experiments and observations of physiologists? Obviously the whole of the circumstances had not previously been taken into account. What may be injurious by itself may be rendered harmless by certain precautions. In my view the essential proviso for the safe administration of nitrogenous food alone is its dilution with copious potations. This point attended to, all danger vanished. The liver and kidneys are enabled to discharge their functions without embarrassment. An active but not excessive metabolism

goes on; the body, in the case of a fat person, lives upon the carbon stored up within it; fat is quickly and continuously absorbed; and it is possible, as many of my statistics show, to bring down the weight as much as three stones in a very few weeks, and to take 12 to 16 inches off abdominal girth. At the same time the health and bodily and mental vigour in suitable cases exhibit marked improvement. I have satisfied myself of this by repeated and almost invariable experience, and my conviction is strengthened by the testimony of others. It happens that amongst my patients there have been many medical men, and the reports which they have furnished during treatment and for some time afterwards are obviously very valuable. One of these, a physician practising in the metropolis, writes under date April 23rd, 1891: "Since commencing your treatment five weeks ago I find that, tested by the spirometer, my breathing power (vital capacity) has increased by 15 to 20 per cent." Another gentleman, who has a large practice in a provincial town, consulted me by letter. In four months he reduced from 17 to 14 stones, and lost 9 inches in abdominal girth. During this period he discharged his duties with energy, and at the end of it writes to say that he seemed no longer disposed to put on fat, and adds: "I think I shall continue the diet for some time longer, as it seems to suit me so remarkably well." It is perhaps a truism to assert that the ordinary functions of the body are better performed by the lean than by the corpulent; but the converse proposition is less apparent, and it is not superfluous to insist upon the fact, which I have often proved, that conditions of ill-health which resist treatment of all other kinds may frequently be made to yield to a strenuous and vigorous diet. As an instance I would quote the following case, originally published by me in the *British Medical Journal*:—

Obesity a Bar to Pregnancy.—Mrs A. consulted the late Dr Matthews Duncan for sterility early in 1890. Married five years; no family. She herself was one of nine children, having three sisters married, all with families. Menstruation irregular and scanty; very obese. Dr Duncan could find no physical reason for sterility except excessive fat. He advised Mrs A. to consult me, which she did on March 10th, 1890. She was placed on dietetic treatment, on the lines laid down and published in the medical journals from time to time since 1887.

The result of treatment was as follows:—*March 10th*, 1890.—Age, 27; weight, 14 stone 5 lbs.; abdominal girth, 52 in.; height, 5 ft. 3 in. *July 15th*, 1890.—Weight, 11 stone 10 lbs.; abdominal girth, 39½ in. In January 1891 Mrs A., after an interval of six months, came to see me, saying she had missed three periods, and had a suspicion she was pregnant. During her treatment menstruation had become regular and healthy. I could not positively, at that stage, say whether she was pregnant or not; however, in due course (Sept. 9th) she was delivered of a healthy boy.

Many well-known writers have pointed out that obesity is a bar to pregnancy. Dr Alexander Duke of Dublin has kindly sent me the *Satellite* (published in Philadelphia) for December 1891, which contains a short paper on "The Influence of Obesity on the Female Sexual Functions," by Dr Juan Rodriguez of Mexico. The remarks are instructive, and the ending more so:—"Sterility caused by obesity may be relieved by removing the cause."

Since what I may term the fundamental principle of my treatment is the strenuous, fearless, and persistent administration of nitrogenous food, I feel called upon to justify this departure at some length. Ample experience, as I have said, has taught me that such a diet is capable of supplying for a considerable time all the needs of the economy. Experience also—the best teacher—enforces the doctrine that the expedient is effective in reducing fat, and the cases I have quoted (which may be supplemented by hundreds more, of which I have notes) go to show that the method is innocuous to ordinary people. There is, however, one class of subjects in whom a special difficulty has been anticipated,—I mean those who suffer from gout. It might be feared that in this case the formation and deposition of uric acid would be much promoted by a nitrogenous diet. Again, I would assert that my experience is against this supposition. Uric acid is no doubt freely formed in some patients, but where this is so it appears to be as freely eliminated.

I am indebted to the courtesy of Sir Alfred Garrod for some researches bearing on this point. From an analysis of the urine of different animals habitually fed on various diets, he obtained the following results:—

	Daily out-put of Uric Acid.
Lion and tiger at the Zoological Gardens,	100,000 per cent.
Man on mixed diet, . . .	10,000 „
Linnet, fed on sugar, seed, and water, .	80 „

Sir Alfred Garrod was also kind enough to furnish me with an analysis, in respect to uric acid, of my own urine, under two different conditions of diet. In the first case I lived freely on a mixed diet of nitrogenous and farinaceous food, drinking also such wines as it is my ordinary habit to take. Under these circumstances the amount of urine passed daily was 70 ounces, and the out-put of uric acid 5·5 grs. per diem. I then dieted myself for three days on nitrogenous food alone, eating 3 lbs. of beef daily and 1 lb. of fish, taking only green vegetables, and drinking spirits and water. The quantity of urine passed in the twenty-four hours was 80 ounces, and the daily out-put of uric acid 10 grs. I believe, then, that in persons whose organs are tolerably sound, the treatment which I advocate may be pursued without injury, and even with benefit, for a much longer time than has been found in any instance necessary. And I would add, that a

weak and encumbered heart is not a contraindication, but rather a further inducement to undertake its relief.

Since the treatment involves some degree of privation, I have devoted all my energies to reducing this to the utmost. It is here above all that I have found the widest scope, the most useful sphere, for innovation in dietetics. Dr Mitchell Bruce says, "The control which we possess over food is the foundation of the vast subject of dietetics." When we consider the extent and importance of the art of cookery alone, and when we further reflect that it has come into existence and developed to meet the requirements of the healthy organism, with the whole range of food stuffs at our service, we must surely admit that its principles are especially applicable to, and its assistance most needed by those on whom restriction is enforced. It is therefore more than ever desirable to consult the preferences of the palate. On this subject Sir William Roberts has written:—"The indications of the palate are of great importance in the regulation of the diet, and should also be inquired into and carefully considered. The palate is placed like a dietetic conscience at the entrance of the gate of food, and its appointed function is to pass summary judgment on the wholesomeness or unwholesomeness of the articles presented to it. It acts under the influence of a natural instinct, which is rarely at fault. This instinct represents an immense accumulation of experience, partly acquired and partly inherited. It is, of course, not infallible,—no instinct is; but so close and true are the sympathies of the palate with the stomach and the rest of the organism, that its dictates are entitled to the utmost deference as those of the rightful authority in the choice of food" (*British Medical Journal*, Oct. 18, 1890). Referring to the unauthorized distrust so commonly found amongst the vulgar, the same writer says:—"This Puritanical view of the palate is wholly unscientific. It, moreover, to speak figuratively, implies a gross slander on a responsible and rarely endowed organ, which has performed in the past, and still performs, most difficult and most complicated functions with complete success; for who shall venture to say that in the evolution of the human animal from the short-lived, immoral, and stupid savage, with his diet of wild fruit, roots, raw flesh, and unfiltered water, to the state of civilized man, the promptings of the palate have not played an important and even an indispensable part?" Applying these excellent maxims to an institution for obesity, it will be found that the palate is at once a trustworthy guide and an exacting master. Never can the Puritanical view alluded to be more fallacious, and till now it has been almost universal. It is this fact more than any other that has wrecked the previous attempts to find a cure for obesity; and further, most of those who have undertaken to treat this troublesome malady have always with diet associated some drug. For my part, I have reckoned with the difficulty from the outset. I have sought to

become acquainted with every variety of available food stuff. I have learnt that this is surprisingly extensive, and by degrees I have provided myself with a frumentarium of very great variety. Compelled to recognise that the taste and requirements of hardly two people are alike, I have learnt by diligent inquiry to know from the ordinary preferences of the patient the best and most agreeable substitute, and in this way I am able to confirm most fully the dictum of Sir William Roberts.

But there are some cases, and they are not always easy to discriminate, in which the forethought, knowledge, and experience—without which, I contend, no one is justified in meddling with obesity—are absolutely needed to guide against disaster. It is an old saying, "What is one man's meat is another man's poison." The regimen which is suited and beneficial to one person may be absolutely injurious to another. I have not met with any untoward events in patients under my own supervision, because I am very careful to obviate mischance, but such have come to my knowledge. It was, unfortunately, a thing of frequent occurrence, more particularly in the earlier days, when I used sets of printed diet cards, which I then thought were suitable to all cases, were passed on by a patient for whom they were intended to others of whom I knew nothing; and so it happened that I unconsciously had treated several people at the same time. Necessarily I am kept unaware in most cases of the process and its result. I suspect that this is sometimes untoward, as it was in the case of a lady who helped herself for advice in this way, but who, unhappily for her, was subject to Bright's disease. I have always pointed out that it is essential that the liver and kidneys, upon which extra work is thrown, should be healthy and capable of bearing the strain. In addition to this, there are peculiarities which must be taken into account and carefully watched. Above all, it should be known by the confiding public that a diet card is not, as a law of the Medes and Persians, immutable, but, on the contrary, indefinitely variable, and that its misappropriation is apt to bring its own penalty.

My original diet charts were printed alike for all cases. I soon, however, found it necessary to vary treatment according to the needs of individuals.

Again, 20-stone cases obviously require more vigorous treatment than more moderate ones. I catechise the patient as to his daily habits, diet, family history as to obesity, and state of general health, and prescribe accordingly.

Again, it is important to curtail, as far as possible, the period of considerable privation. From the knowledge that there is nothing to fear from the energetic administration of nitrogenous food, it happily results this may be done safely in the most rational manner. The first fortnight suffices for the complete degree of privation; even that in certain cases may be greatly modified by permission to use a considerable selection of vegetables, and the

necessity of taking fluid abundantly enables me to propitiate the palate, and to combat nausea by the exhibition of stimulants, aërated waters, and light wines to almost any desired extent. The course of treatment is divided into three periods. Of the first sufficient has been said. In the second and third restrictions are gradually removed. After the lapse of nine weeks the patient may return to his usual mode of life. Let that be in the words of Prince Hal to Falstaff,—"Drinking of old sack, unbuttoning him after supper, and sleeping upon the benches after noon." Even then he will have a long start of his foe, and he may devote many months and even years to storing up fat before he will have regained the two or three stones lost so easily and in so short a term. Indeed, I have most commonly found, as in the case of the medical man already quoted, that hardly any tendency to obesity remains; but I advise a return to a restricted diet at intervals of two or three months for a period of ten days, which I have found amply sufficient to prevent any serious increase of bulk or weight, but it is necessary to prohibit permanently the use of sugar and beer. I am well aware that a method of treatment such as this will be judged by results. That is the criterion which I have myself applied to it. I have published the results of forty-two cases in the *British Medical Journal* of 10th November 1888, and of thirty-two cases in the issue of 31st May 1890. I would refer to these, and add also to them statistics of cases that have come under treatment up to date.

Case 1.

Sex.	Age.	Height.	Weight.	Abdominal girth.	Date.
M.	60	5 ft. 4½ in.	13 st. 4½ lbs.	46 inches.	20/4/91
...	...	...	11 st. 6¼ lbs.	37 inches.	15/7/91

This gentleman, a member of the profession, having tried every means of reduction, consulted me, and obtained the above result. His heart and lungs were both affected, and it was not uncommon for him to have attacks of syncope. Since his treatment he walks four and five miles a day, and enjoys perfect health.

Case 2.

Sex.	Age.	Height.	Weight.	Abdominal girth.	Date.
F.	39	5 ft. 3 in.	15 st. 9 lbs.	58 inches.	25/9/91
...	...	...	13 st. 3 lbs.	47 inches.	26/1/92

Greatly improved in general health.

Case 3.

Sex.	Age	Height.	Weight.	Abdominal girth.	Date.
M.	35	6 ft.	17 st. 8½ lbs.	50 inches.	16/10/91
...	...	...	13 st. 13½ lbs.	41 inches.	10/3/92

Mr Daniell of Blandford says this gentleman has never been so well or fit in his life.

Case 4.

Sex.	Age.	Height.	Weight.	Abdominal girth.	Date.
F.	27	5 ft. 1 in.	12 st. 9¾ lbs.	48½ inches.	10/12/91
...	...	...	10 st. 6 lbs.	40 inches.	25/1/92

Dr Griffiths of Harley Street, finding this patient extremely obese and suffering from attacks of syncope three or four times a week, suggested treatment for fat. Under this the attacks of syncope have entirely vanished.

As showing the amount of loss that may be sustained by exercise, a friend, a medical practitioner, cycled one hundred miles in ten hours. Commencing on an empty stomach, and taking during the journey *a small* quantity of bread and cheese with milk and soda-water. The weather was extremely hot, and perspiration very excessive. Weight on starting, 11 st. 7 lbs.; on completion of the journey in ten hours, weight 10 st. 9 lbs., a loss of 12 lbs. He goes on to say that he was not very tired, but was extremely shaky and nervous, and so much altered in physiognomy that his immediate family scarcely recognised him.

I have sufficiently indicated the circumstances to which I attribute my success. I have made sufficiently prominent the necessity of limiting, as far as possible, the privation imposed and the necessity of studying and consulting the patient's state. But this will be useless without a sufficient knowledge of the means by which, and the limits within which it may be safely gratified. This knowledge can be obtained only by patient and careful inquiries; and he who would treat obesity with success must be prepared with resources which are practically unlimited.

Reverting to the general consideration with which I prefaced my remarks, I would insist that as medical practitioners whose lot is cast amongst a people overburdened with wealth, inheriting predispositions which are often wholly at variance with individual habits, the latter day reproduction, in many of their conditions, of whose bodily weaknesses have brought about the downfall of great nations and have spread some of the saddest phases of history, we hold in our hand a weapon powerful for much good; we have imposed upon us by our knowledge a new duty, and we must not be tempted to pass it by.

Diet for an Extreme Case.

1*st Period,* 14 *Days.*

Breakfast.—Tea or coffee, with saccharin if desired in lieu of sugar; bread or biscuits made from soya bean, 2 oz.; grilled white fish or red meat, kidneys.

Lunch.—Cut from joint of beef or mutton, taking no fat, and one helping of green vegetables, avoiding only peas, beans, and all roots; soya bread or biscuit, 1 oz.

Dinner.—Clear soup, white fish, red meat, green vegetables as at lunch; soya bread or biscuit, 1 oz.

Drink.

First thing on waking.—Tumbler of hot water with slice of lemon.

11 A.M.—Cup of bovril or clear soup.

Lunch.—Two glasses of claret or 1 oz. of whisky with potash water.

5 P.M.—Cup of bovril or clear soup.

Dinner.—Two glasses of still hock or claret, or whisky and potash.

Bedtime.—Cup of bovril or clear soup.

Mustard, pepper, salt, Harvey's sauce, may be taken.

2nd Period, 21 *Days.*

Additions to No. 1.—Oysters, tongue, stewed fruit, with saccharin; poultry, game.

3rd Period, 31 *Days.*

Additions to No. 2.—Toast in place of soya bread, for each meal, 2 oz., savoury jellies, aspic of prawns, plovers' eggs, jelly.

Desert.—A small quantity of fruit; blue-mould Dorset cheese.

Suppose a case, 20 stones, requiring reduction 4 stones. The above diet will, provided a large amount of liquid be taken in the 66 days, be sufficient. A return to usual diet may be resumed. The rise will be gradual, and 10 days' resumption of either No. 1 or 2 periods, three or four times per annum, will be amply sufficient to regulate weight and bulk in future.

A moderate amount of exercise proves useful. In dealing with milder cases, commence with No. 2, or even 3, when an equally satisfactory result may be obtained.

Hours of meals must be regulated by usual habits of each patient, and it may be also useful to say that nearly all cases require variation of rules as to diet, though the principle remains the same.

No. 1.—SPECIMEN DIET CHART.

This diet sheet is arranged in accordance with usual habits and family history as to obesity, and must be strictly confined to personal use.

Name and address.....189

Diet Chart for 14 *Days.*

7 A.M.—Sip slowly a tumbler of hot water with lemon juice.

9 A.M.—*Breakfast.*—Two cups of tea or coffee without sugar or milk, taking saccharin if needed; 1 oz. of soya bread or biscuit; grilled white fish, steak, chop, kidneys.
11 A.M.—Tumbler of hot bovril or clear soup.
1.30 P.M.—*Lunch.*—Cut from joint of beef or mutton, with one helping of either cabbage, spinach, tomatoes, asparagus, French beans, plain lettuce, and watercress; 1 oz. of soya bread or biscuit.
5 P.M.—*Afternoon Tea.*—Cup of tea *a là Russe,* or cup of bovril.
7.30 „ —*Dinner.*—Clear soup, white fish, red meat, vegetables as at lunch; 1 oz. of soya bread or biscuit.
Bedtime.—Tumbler of hot bovril or clear soup.

1. All food should be plainly cooked (grilled for preference), no fat, skin, or rich *gravy* should be taken.
2. Drink claret, still hock, burgundy, Scotch whisky, and potash water.
3. Exercise—a moderate amount of walking should be done daily.
4. Condiments—Mushroom ketchup, Worcester and anchovy sauce, mustard, pepper, and salt may be taken.

The President hoped that the paper might elicit discussion on the subject.

Dr James Ritchie asked whether Dr Towers-Smith meant literally that the weight should be reduced as rapidly as possible. The speaker had found evil effects from a very rapid reduction, and guarded his patients accordingly. Dr Towers-Smith recommended a purely nitrogenous diet—did he mean this absolutely, and did he get his patients to carry out his recommendations for any prolonged period of time? Although dieting is a very important matter, nevertheless some people have such a tendency to the deposition of fat, even when put upon spare diet, that other methods were important. Dr Towers-Smith had told us the kinds of alcoholics which he recommends. Dr Ritchie considered alcohol prejudicial in obesity, because it interfered with oxidation and favoured the deposition of fat. Stout people are usually thirsty beings, and the speaker agreed with Dr Towers-Smith that a considerable quantity of water was essential to them in order to favour the elimination of waste products, but as large quantities of fluid at wrong times favoured the production of fat, did he lay down any rule as to the times at which fluid should be taken? Dr Ritchie recommended little fluid at meals, but a fair quantity three hours afterwards. What regulations as to exercise were recommended? Were forty winks after meals proscribed?

Dr Hunter said they were indebted to Dr Towers-Smith for bringing his paper before the Society, but he would have conferred greater obligation had he gone into more details regarding his diet tables, *e.g.*, what were the varieties of nitrogenous foods he allowed

for breakfast, lunch, and dinner. It would also have been satisfactory to have had some rational or scientific brief explanation of his dietary on physiological principles. Dr Towers-Smith had stated that the proper proportion of nitrogen to carbon in foods was given in works on dietetics as 1 to 4, but it is pretty generally agreed that the ratio is as high as 1 to 15. Physiologists knew that life could be maintained satisfactorily for a considerable time on water and nitrogenous food alone, and the explanation was, that a considerable proportion of the latter during its tissue metabolism became converted into fat. This really meant that the plan of diet recommended by Dr Smith was a modified starvation one, for the carbonaceous food had to be derived from the proteids, and the latter must therefore *pari passu* be diminished.

Dr Church regretted that so little reference had been made to the subject of fresh air and exercise in the paper. Some people who took very little food, and many who took a well-proportioned amount of nitrogenous and non-nitrogenous food, but who, by deficient exercise in the fresh air had not their blood sufficiently oxygenated, suffered from obesity. Dieting alone without due regulation of exercise out of doors could not healthily reduce obesity. Indeed, exercise must go hand and hand with dieting in the treatment of the condition.

Dr Boyd, in adding his thanks to those of other members, would like to ask a question. Dr Towers-Smith quotes some experiments on diet where the nitrogenous excretion was estimated by the amount of the uric acid which was passed. Now, one would like to know how much nitrogen was being taken by the food? How much exercise was being taken? How much nitrogen was being excreted by the bowel? Without these data, Dr Boyd did not think that any conclusions could be drawn from the experiments.

Mr Cotterill asked Dr Towers-Smith if he could explain how it was that there was no inclination to a return to obesity after his treatment, when in the case of athletes, who reduce their weight by a somewhat similar process, there is usually a rapid return to the normal weight.

Dr W. Towers-Smith said, in reply to Dr James Ritchie, that with regard to the rapid loss of weight the essence of his system of dietetic treatment consisted in the rapid reduction with safety. No doubt it was better to avoid alcohol if possible. He had found that the least hurtful form was Scotch whisky. If a lean and a stout man took exercise, the stout man excreted by the skin more, and required fluid. As the diet consisted in the earlier stages principally of nitrogenous material, a large amount of fluid was required. Dr Hunter had suggested details of the diet, but no two people were treated alike. There was no starvation possible, as the amount of food ordered was unlimited. Nitrogenous food did not make fat, a destructive metabolism went on, and so a large amount of fluid assisted the carrying off of the burnt

carbon. In reply to Dr Church, a moderate amount of daily exercise is necessary—walking, riding, cycling, lawn-tennis. All these are ordered, but not to be overdone. In answer to Mr Cotterill, Dr Towers-Smith said, so soon as a proper amount of reduction of bulk and weight is attained, the patients may safely resume their ordinary, barring sugar and beer—abstention from these must be permanent. The rise in weight is very gradual—about 3 to 4 lbs. a month being the average. I provide patients with a restricted diet to follow for 10 days three or four times per annum. I have found this amply sufficient to keep people right.

Meeting VIII.—April 6, 1892.

Mr Joseph Bell, *President, in the Chair.*

I. Exhibition of Patients.

1. *Dr J. W. Martin* showed three cases of PHTHISIS which had been cured. The first had had all the symptoms of phthisis—night sweats, cough, spit, hæmoptysis, emaciation, etc. There were signs of consolidation in the chest. Under diet, counter irritation of the chest, inhalations, and internal medication, the patient gained 1½ stones in weight, and was now apparently in perfect health. The second was a case not quite so far advanced, which under similar treatment had made a good recovery. The third, a case in which the febrile symptoms were very marked, with all the signs of phthisis with cavity formations, and in which bed sores formed, became convalescent under similar treatment, and was now able to follow his occupation.

2. *Dr Shand* showed a patient on whom AMPUTATION OF THE THIGH UNDER ICE had been performed thirty years ago. Union took place upon the third day by first intention. No other antiseptic was used.

3. *Dr Allan Jamieson* exhibited—(*a.*) A case of DERMATITIS PAPILLARIS CAPILLITII, OR ACNE KELOID. He remarked—Under the first of these terms, Kaposi in 1869 described an inflammatory condition of the hair system, seated particularly at the nape of the neck, but invading the occiput or being limited to the crown, allied to sycosis, but leading to changes of a peculiar type, which are well exemplified in the patient now shown. It will be seen that these affect the beard and not the scalp, and it is now known that Kaposi was in error in restricting the localization to the scalp, as it may affect any hairy part. The patient, now aged thirty-two, and in otherwise good health, first came under my care two years since, and had then suffered from the disease for eighteen months. It com-

menced by the formation of a pustule in the beard, pretty well down on the side of the neck on the left side. More formed, and produced a thickly crusted area, which itched a good deal. After a month's treatment he considerably improved, and I lost sight of him till a fortnight since. Then thick crusts concealed the true state of matters. These were removed by boracic starch poultices. You will observe that on the left side of the neck, the beard, elsewhere close and healthy, is for a space of 4 inches across by 2 deep replaced by a hard, gristly, almost immovable formation, nodular and uneven. On this the hairs, dark and remarkably strong in contrast to the much fairer and softer ones of the rest of the beard, are grouped into bundles like the bristles of a brush, imparting a most extraordinary appearance to the affected part. When the hairs are forcibly extracted they can be removed only with much difficulty and pain, and are seen to be deeply and very firmly implanted. Leloir and Vidal regard this as a pilo-sebaceous folliculitis, with a tendency to the formation of keloid. Dubreuilh shows that it commences in the sebaceous gland, leading to an abscess in the neighbourhood of the hair follicle. There is a perifolliculitis followed by keloid. Keloid, it is well known, often starts in acne scars and extends from this point of origin. Here it immediately succeeds the acne, the adjoining follicles are infected, and eventually the gristly uneven tissue here seen is produced. Great benefit has resulted from washing with superfatted potash soap, and the subsequent inunction of an ointment of sulphur and ammoniated mercury. As to the cause little can at present be said. Certain persons and certain races develop keloid much more readily than others; there is a predisposition in this direction, but whether this is set agoing by microbial agency or not is quite undecided. (*b.*) Case of MUCOUS PATCH, OR CONDYLOMA, apparently due to trichophyton. John M., aged 20 months, was brought by his mother, a remarkably healthy-looking woman, to the Royal Infirmary on March 5th, 1892. No history or suspicion of specific taint could be elicited by careful questioning, either on the father's or mother's side. The child itself was well grown, and past the age at which usually any evidences of inherited syphilis of the secondary era are still persistent. It appeared that in the beginning of January a small round patch showed itself on the right shoulder, which was pronounced ringworm by their doctor, and shortly disappeared when painted with iodine. Soon after this a little pinkish elevation was noticed in the cleft of the nates close to the margin of the anus, on the right side. This grew slowly at first, but had in course of the two previous weeks increased to the size of a shilling, being divided transversely by a fissure, and being surrounded by an areola rather more than an eighth of an inch broad. It resembled in the most exact manner an ordinary condyloma. Some of the epithelium on its surface was scraped off and examined for trichophyton, but no

mycelium nor spores could be seen. There were also three or four vesico-pustules on the left buttock. Washing daily with superfatted potash soap and warm water, followed by inunction with a sulphur and ammoniated mercury ointment, has caused its complete disappearance. Some time since an almost exactly similar growth was seen inside one nostril of a healthy boy from the country, who worked among cattle, and which was cured by like treatment. Both situations are unique ones for ringworm, and though microscopic verification is awanting, there is strong presumptive evidence that this was a case of ringworm in an unusual situation. The heat and maceration on the tender skin of the infant had the same effect on the patch of ringworm that it has on the flat syphilitic papule. At the same time, in one case of inherited syphilis a condyloma at the margin of the anus was the sole remaining trace of syphilis in a child aged twenty-one months. In it, however, there was a clear history of hereditary disease. Brocq has met with an indisputable case in the person of a young man of lymphatic constitution, free from all venereal taint, affected with eczema at the verge of the anus. He mentions also that they may be met with in pregnant women who have copious leucorrhœa.

4. *Mr Caird* showed two cases, in women, of THYROIDECTOMY. The first had the right lobe and portion of the isthmus removed on account of a rapidly growing goitre which had interfered with respiration and deglutition. The operation was carried out fourteen days ago. The wound had healed by first intention, and the patient getting out of bed on the ninth day. All respiratory embarrassment had disappeared. There was still slight dysphagia. The second suffered from EXOPHTHALMIC GOITRE. Removal of the right lobe was effected about a year ago with singularly beneficial result. During the last few months, however, symptoms of headache and palpitation had re-developed, but there was no increased proptosis.

II. Original Communications.

1. NOTES ON A CASE OF PAROXYSMAL METHÆMOGLOBINURIA.

By A. Lockhart Gillespie, M.B., M.R.C.P. Ed., Medical Registrar, Edinburgh Royal Infirmary.

Last winter Dr Muirhead kindly asked me to examine a case of paroxysmal hæmoglobinuria, which had been lately admitted into Ward 29. I have now the pleasure of laying some notes of the case before the Society.

George M'V., aged 26, married, a hawker, was admitted into the Edinburgh Royal Infirmary on November 25th, 1890, complaining that his urine became at times very dark coloured. With a precarious employment, a poor home, often too scanty a

supply of food, his experiences of life had not been of the happiest; he, however, had not indulged overmuch in liquor, and had enjoyed reasonably good health up to two years before admission into hospital. About that time he noticed that he became more prone to take cold; that if he were chilled in any way he was seized with a rigor; his urine, however, was normal as yet. These attacks grew more and more severe, and were accompanied by the passage, for a few hours after an attack, of dark porter-coloured urine. His description of a severe attack was as follows:—On going out on a cold day he would suddenly become chilled all over, benumbed, with acute pain in the stomach region, so acute sometimes as to double him up; he would then eructate much wind, with great relief to himself. During the attack he felt giddy, but never fell, while his testicles were retracted and his skin muscles contracted, causing a condition of goose-skin. There never was any pain over the kidneys.

His first severe attack occurred when he was watching some races at Powderhall on a very cold day,—he was not sure of the date, probably in spring 1890,—while since then he has passed blood in the urine once or twice every week. He does not appear to have had syphilis, nor has he ever been abroad. There was no correspondence, as far as I could ascertain, between drink and the disease.

Condition on admission.—A very small, badly-developed man, miserably nourished, height 4 ft. 10¾ in., weight 6st. 9 lbs., obviously anæmic. Physical examination revealed no abnormality, his tongue was a little flabby and furred, his bowels slightly costive. Analysis of the urine on admission gave the specific gravity as 1024, reaction acid, no albumen or sugar, urea 7·4 grs. per ounce, or the amount of urine being 36 ounces in the twenty-four hours, 266·4 grs. on a hospital diet. Temperature normal.

Progress.—The day after admission he was sent out for a walk; the weather happened, as usual, to be bad, and he came in with a temperature of 100° Fahr., reporting that he had not been long out before he began to shiver. On his return he passed some very dark urine. It was at this stage that I was asked to examine the case more carefully, and was given a sample of the dark urine passed the day before for analysis. As the urine passed during the attack had been mixed with the urine previously passed, I contented myself here with a determination of the pigment. On examining it with the spectroscope, I found the four bands which are characteristic of acid hæmatin, and which further were not reduced to the one broad band of reduced hæmoglobin on the addition of a small quantity of ammonium sulphide. There were no blood corpuscles or casts, and the fluid, which was faintly acid, deposited only a small quantity of dark granular debris.

For six days after this attack the patient was kept quietly in bed, and was given 3j. doses of the liquid extract of ergot every eight hours. On the sixth day I tested his urine and examined his blood.

The urine was 1016, acidity ·1008 per cent. (reckoned as HCl), no albumen, sugar, or blood, urea 107·5 grs., no deposit; while the blood contained 3,100,000 red corpuscles, which were irregular, and crenated, some having vacuoles in them. The white corpuscles, 100,000, were above the normal. The amount of hæmoglobin present was 65 per cent., that is, the proportion per corpuscle was $\frac{65}{62}$, or 104·8 per cent. On the 4th of December, two days later, the examination of the blood showed that the number of red corpuscles had increased to 3,370,000; the hæmoglobin was 63 per cent., or 93·4 per cent. per corpuscle. The pigment present was oxyhæmoglobin, the red corpuscles were from 6 to 8 μ in breadth, no very small ones, but some tailed and others crenated. White corpuscles 20,000. Plates numerous.

The urine for the day before contained 8·4093 grs. of urea per oz., or 319·55 grs. per diem, had a specific gravity of 1021, a deposit of mucus and oxalates, and an acidity of ·18 per cent. HCl, or ·06624 gram. of HCl in the day.

The urine of the 4th contained 5·8325 grs. of urea per oz., or 225·3 grs. altogether, the specific gravity was 1020, acidity ·054 per cent., or ·0216 gram. of HCl per diem; there was a deposit of phosphates and mucus.

On the 5th he was allowed up and about the ward, and on the 6th the red corpuscles numbered 3,080,000, the white 10,000, the plates were not so numerous, while the red corpuscles, measuring from 6 to 10 μ, were larger, and were neither tailed nor crenated. The hæmoglobin was 63 per cent., or 103·2 per corpuscle. Urine: sp. gr., 1020; acidity, ·126 per cent., or ·05796 gram. per diem; urea, 7·0726 grs. per oz., or 325·33 grs. Oxalates present; no albumen or blood.

7th December.—He was up all day yesterday. He had some slight diarrhœa, his bowels moving thrice. The urine was more concentrated; sp. gr., 1024; acidity, ·216 per cent., or ·07344 gram.; urea, 12·221 grs. per oz., or 415·514 grs. per diem.

8th December.—Up all yesterday, but not allowed out. Blood analysis shows the number of red corpuscles to be 3,990,000, the hæmoglobin 55 per cent.; proportion per corpuscle, 68 per cent.; white corpuscles 12,000; plates more numerous. The red corpuscles were from 7 to 10 μ in size, some were mere phantoms with only the outer envelope, and were discoloured, but of full size; one or two corpuscles were tailed, none crenated. Urine: sp. gr., 1020; acidity, ·108 per cent., or ·05832 gram.; urea 6·911 grs. per oz., or 373·194 grs. per diem. No albumen or blood.

9th December.—Urine: sp. gr. 1020; acidity, ·144 per cent., or ·06048 gram.; urea, 7·0972 grs. per oz., or 298·0824 grs. per diem. Patient had been out for a short time the day before, but did not feel cold; temperature, 54° Fahr.

10th December.—Yesterday he was sent out for most of the day somewhat thinly clad; on coming home he passed a few drops of

dark-coloured urine, which were unfortunately mixed with the day's total. He neither felt the shivering nor the malaise which had formerly occurred. His temperature rose in the evening. Blood analysis: Red corpuscles, 2,200,000, from 4 to 7 μ in size; many more were irregular than in the last counting; none, however, tailed or crenated. No large ones seen, but many (about 60,000) small, colourless, ill-defined corpuscles, which were neither granular nor refracting. No pigment granules seen. Hæmoglobin, 70 per cent., or 159 per cent. per corpuscle. Spectrum gives only the oxyhæmoglobin bands. Urine, sp. gr. 1022, with a deposit of very small oxalate of lime crystals. Albumen and blood present. The pigment was very dilute, and gave the bands of oxyhæmoglobin, and more faintly those of methæmoglobin. These bands were reduced on the addition of ammonium sulphide. The total proteids were only ·108 per cent., of which the serum albumen formed ·025 per cent., the serum globulin ·083 per cent. The urea, 7·0513 grs. per oz., came to 253·84 grs. in the day; the acidity, ·108 per cent., or ·03888 gram. The liver had been mapped out before, and showed no change; but the spleen was enlarged downwards and forwards, coming forward nearly as far as the nipple line at the level of the eighth or ninth ribs. There was no tenderness.

12*th December*.—He was out again for a short time yesterday, but had no attack. To-day the urine is clear; no blood or albumen; sp. gr., 1017; urea, 6·3054 grs. per oz., or 290·048 grs. per diem; acidity, ·108 per cent. HCl, or ·04968 gram. Blood analysis: Red corpuscles, 3,530,000, size 6 to 9 μ; some tailed, not so irregular as yesterday. White corpuscles, 15,000. Plates as usual. Hæmoglobin, 66 per cent., or per corpuscle 93·4 per cent. The spleen, if anything, is smaller than yesterday.

15*th December*.—Yesterday the day was colder, and although only out about three hours he felt very cold; he did not shiver, however. Temperature up; urine dark red. Blood analysis: Red corpuscles, 2,980,000, of various sizes, from 5 to 9 μ; none tailed or vacuolated, but one or two very shadowy. White corpuscles, 20,000. Plates numerous. Hæmoglobin, 64 per cent., or 107·3 per cent. Urine the colour of port wine, clear, no deposit; sp. gr., 1031; no sugar; acidity, ·162 per cent., or ·08748 gram. per diem; urea, 9·524 grs. per oz., or 514·296 grs. altogether. The pigments present in the urine were oxyhæmoglobin and a trace of methæmoglobin, the lines distinctive of the latter only being visible in a thick layer of the fluid. The proteids present were ·6 per cent., of which serum albumen formed ·25 per cent., the globulin ·35 per cent. His spleen had again enlarged, but was not tender.

On the 16th the hæmoglobin had risen to 70 per cent., the red corpuscles to 3,850,000, or 90·9 per cent. per corpuscle. One or two phantom corpuscles, while the red cells as a rule were smaller

but more regular, 4 to 6 μ in size, forming fairly good rouleaux. Urine: pale, deposit of mucus; sp. gr., 1025; urea, 232·96 grs. in the day; acid, ·03024 gram. No albumen or blood.

Seeing that his blood was in such an unstable condition, I resolved to try the effect of sulphonal on it, so I gave him 20 grs. on the night of the 16th. On the 17th his blood contained 2,930,000 red corpuscles, many of which were phantom; size, 5 to 8 μ; none crenated, many elongated. Hæmoglobin, 70 per cent., or 119 per cent. per corpuscle. Urine contained no albumen or uro-hæmato-porphyrin, and was normal.

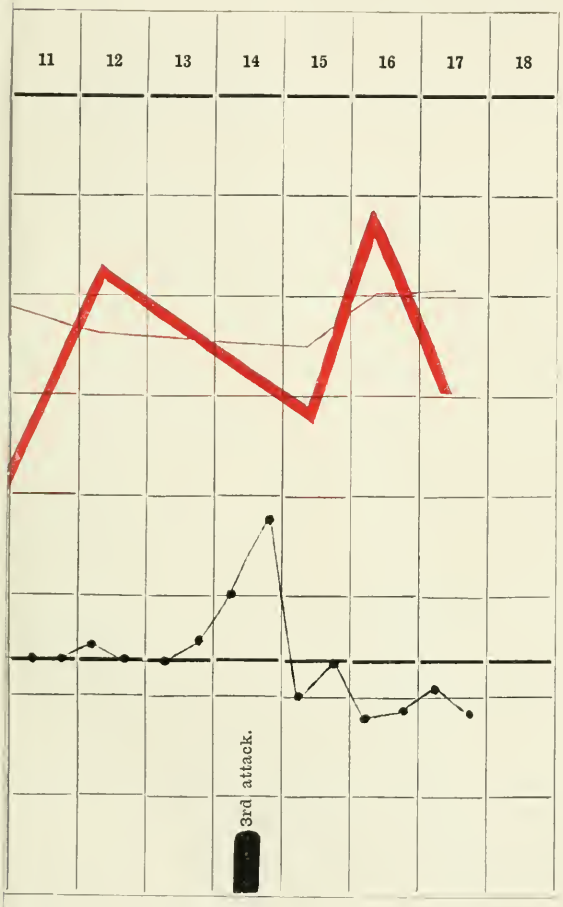
I gave him 30 grs. of sulphonal that night again, with no discoloration in the urine, and 35 grs. on the following night, with a similar want of success. He now expressed a desire to go home, so that I had to stop my observations. On dismissal he felt and looked very much better.

Conclusions.

The points I would particularly draw attention to are—first, the enlargement of the spleen with each attack; second, the fact that the percentage of hæmoglobin did not fall *pari passu* with the corpuscles; third, that the total urea excreted during the day increased on the day of the attack, contrary to what is otherwise affirmed (*Münchener Medicinischer Wochenschrift*, 1888, xxxv., pp. 495, 529, and 535). The proteids, too, merit attention, as does the temperature. The size of the corpuscles, also, varies after the attacks.

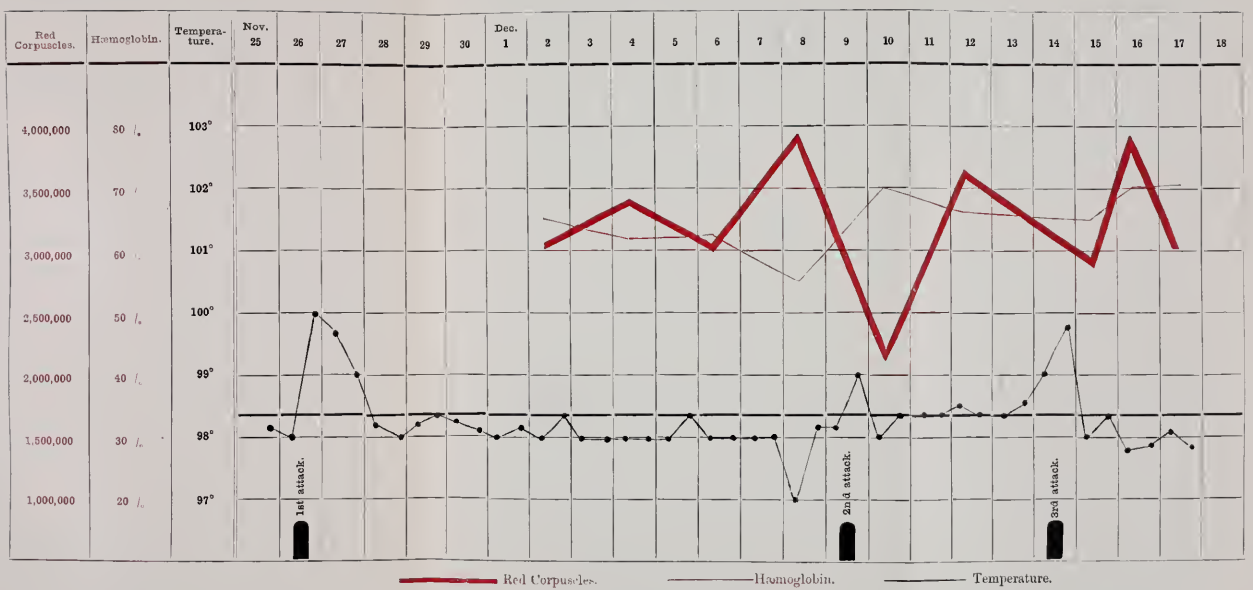
The enlargement of the spleen, which occurred in this case, was coincident with the formation of numbers of young red blood-corpuscles.

Paroxysmal hæmoglobinuria is a local disease and yet a general one. The breaking down of the red corpuscles and consequent setting free of hæmoglobin occurs in the blood. This is shown conclusively by Boas' experiment, repeated by Copeman, of tying a ligature tightly round a finger, then dipping the finger into ice-cold water. Examination of the blood of this finger now shows free pigment in the plasma, with a loss of the power, inherent in the red cells, of forming rouleaux. There is little or no general disturbance. If both the hands, however, are placed in iced water for a short time, there is a decided disturbance, and pigment may or may not appear in the urine. Again, it has been pointed out that if a small quantity of hæmoglobin be injected into the blood of a healthy individual, none of it appears in the urine; if a little more be injected, some albumen may make its appearance; but on injecting large quantities, analysis of the urine shows the presence both of hæmoglobin and albumen. So in a case like this, if the disturbance be great and the tale of corpuscles destroyed large, the urine becomes porter-coloured. If the exciting cause be not so severe, only serum albumen and globulin



——— Temperature.

CHART I.


Red Corpuscles.
Hæmoglobin.
Temperature.
Nov. 25
26
27
28
29
30
Dec. 1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
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18
4,000,000
3,500,000
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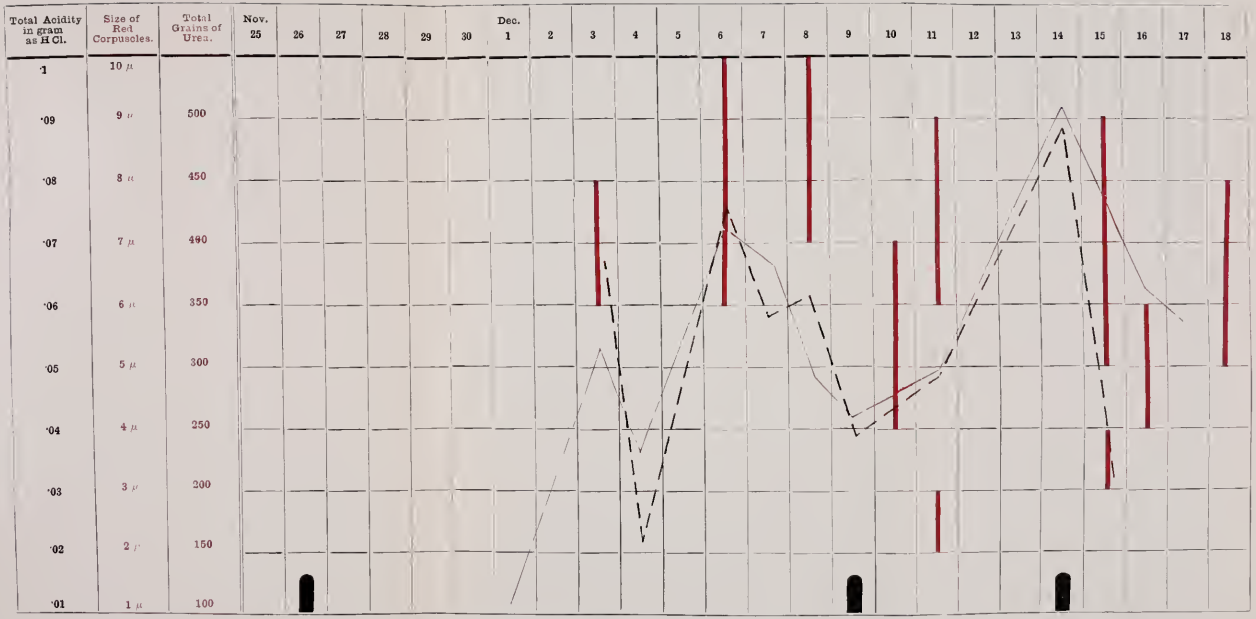
1,500,000
1,000,000
80 /.
70 /
60 /.
50 /.
40 /.
30 /.
20 /.
103°
102°
101°
100°
99°
98°
97°
1st attack.
2nd attack.
3rd attack.
Red Corpuscles.
Hæmoglobin.
Temperature.

es.

CHART II.



The red line denotes total grains of urea per diem.
The perpendicular red lines are intended to represent the variations in the size of the red corpuscles.
The broken black line represents the total acidity of the urine in grams of H Cl.
The thick black marks indicate the attacks.

may appear, constituting paroxysmal albuminuria. While on other occasions I believe you may get a slight attack with no striking evidences of it in the urine, a slight rise of temperature and diminution of red cells in the blood would be the only symptoms, along with a slight excess in the normal pigments of the urine. That is to say, then, that the organs of the body—principally, of course, the liver—can get rid of a certain quantity of free hæmoglobin; if the blood, however, be surcharged with it, it is eliminated by the kidney. Whenever sufficient has been excreted to allow the liver to deal with it in the ordinary way, the discharge in the urine ceases.

I will not enter into the subject of the elimination of proteids in the urine without coincident disease of the kidney tissue. It opens too wide a field. Selective affinity, and perhaps change and increase of blood-pressure, may partly account for it. In this case the serum globulin was in excess in the blood and in the urine. In ordinary cases of Bright's disease, the serum albumen is in excess. Excess of globulin over albumen in the urine is said to be due to increased blood-pressure.

Red blood-corpuscles are now formed in great numbers to make up for the deficiency caused by the previous destruction; and if the observation that the spleen enlarges after the attacks is confirmed, it is good evidence that the manufactory of the young cells may take place there.

The variation in the size of the red corpuscles is very instructive, apart from the mere irregularity and vacuolation of many of the larger ones. On reference to Chart II., it will be seen that after the second attack the corpuscles varied from 4 to 7 μ on the day after, and on the next day from 2 to 3 μ and from 6 to 9 μ. After the third attack the sizes were from 2·5 to 9 μ. At the end of the considerable period that elapsed between the first and the second attack, the red corpuscles measured from 7 to 10 μ, or about the normal.

I tested the acidity of the urine each time, but with no result, save that as the urea increased so did the acid. Bearing in mind, however, the inverse proportion which always maintains between the acid eliminated by the kidneys and that present in the gastric juice, there is presumably a deficiency of acid in the stomach contents during and for some hours after an attack. This may account for the gastric symptoms detailed above.

With regard to the pigment present, on one occasion I found acid hæmatin, on three occasions methæmoglobin and oxyhæmoglobin, and in two other cases which I have seen, but which I had not an opportunity to examine further, the pigment was methæmoglobin. Copeman explains the presence of acid hæmatin by the action of the acid urine on the methæmoglobin, and asserts that the pigment, as originally secreted by the kidneys, is in the form of oxyhæmoglobin; this soon changes into methæmoglobin

in the urine, and if left long enough, into acid hæmatin. Certainly after one of the attacks the urine I examined contained the latter pigment, as sulphide of ammonium had no effect on it, the four bands in the spectrum remaining as before. M'Munn, in 1889, asserted that methæmoglobin was the only pigment he had ever found in such cases. It is also certain, however, that the blood serum itself during an attack is stained not with methæmoglobin, but with oxyhæmoglobin.

The effect of nitrites and of chlorate of potash in inducing attacks has been tried in experiments with negative result.

About the time I was investigating the case, some attention was drawn to the appearance of uro-hæmato-porphyrin in the urine, following on the exhibition of sulphonal and allied hypnotics in large doses. Desirous of testing the effect of this drug on the evidently unstable corpuscles of this patient, I gave him, as recorded above, considerable doses of it each night, but with negative results. The patient, indeed, thought fit to end the treatment by abruptly leaving the hospital. The paper by Copeman in the *Practitioner* for 1890, p. 161, contains a fairly complete bibliography.

The analyses for the above were made at the Laboratory of the Royal College of Physicians, Edinburgh.

The President congratulated the Society on having such a scientific and practical paper read to them, and expressed his hope that a valuable discussion might result.

Dr Allan Jamieson had nothing to say except of a complimentary nature. The use Dr Gillespie had made of the spectroscope was interesting, and, if continued, might lead to further discoveries.

Dr J. W. Martin said he thought it was peculiar in this case, as the patient had not been in a malarious country. Was this a case of undiagnosed ague? In this case the urine was much darker than in the malarial hæmaturia. There was some connexion with the spleen in this case. More careful and more frequent measurements of the spleen might have been made. He had not tried the effect of a blister on the surface. The case was of interest from its rarity as not being due to parasites. It must have a bearing on the sympathetic functions of the spleen and the kidney, and perhaps liver.

Dr Gillespie thanked the Society for the compliments paid him by those who had spoken. He pointed out that it was a purely scientific inquiry, not a clinical one. He had not used pyrogallic acid to endeavour to produce an attack. The investigation only covered a short period, and was not intended to be comprehensive.

2. OBSERVATIONS ON THE ACTION OF ANTIMONY IN DISEASES OF THE SKIN.

By W. Allan Jamieson, M.D., F.R.C.P. Ed., Physician for Diseases of the Skin, Royal Infirmary; Lecturer on Diseases of the Skin, Edinburgh School of Medicine; and A. Home Douglas, M.B., F.R.C.P. Ed.

The results obtained from the administration of tartarized antimony in some rather intractable affections of the skin, in a series of cases already published by one of us,[1] encouraged a further trial of the remedy under conditions which admitted of a more careful study of its action, with the object of determining, as far as possible from clinical facts, its mode of operation, and of perhaps affording more precise indications for its successful employment. The following investigations have been carried out in the recently established wards for Diseases of the Skin in the Royal Infirmary.

Antimony had, after being too extravagantly praised, fallen much into desuetude, in an exactly similar manner, though to a greater degree than mercury, which was distrusted at a not very distant period as a remedy for syphilis, a result which surely followed from the crude and unscientific mode in which it had been till recently employed. Another reason for the comparative neglect of antimony may possibly be found explained in that study of the natural course of disease, so persistently advocated by the late Professor Hughes Bennett, which the revulsion against the routine and heroic use of drugs had brought so prominently forward, and which has since yielded such valuable conclusions. Hence the statements in the text-books both of medicine and therapeutics are, to a large extent, not based on observations of very modern date. Thus among other points careful records of the temperature, in cases where antimony has been given for long periods, are deficient. Skin diseases offer an unequalled field for testing the value of such a powerful drug, but before proceeding to relate the cases we have followed, or to formulate the inferences we have drawn, it is necessary briefly to refer to the current opinions which are held as to the action of antimony on the system. In none of the works which we have consulted have we found these better summarized than in those of Dr Lauder Brunton and Dr Mitchell Bruce. It is there laid down that it influences metabolism, causing fatty degeneration of the organs, and increase of the nitrogenous products, oxygenation being comparatively deficient. It is regarded as a general depressant, lowering the circulatory, respiratory, and nervous systems. Among other modes in which it leaves the body, it is believed to be excreted partly by the skin, in the perspiration. "It is found to be a powerful general depressant, oxygenation being impaired, nervo-muscular activity

[1] "On the Value of Antimony in various Inflammatory Affections of the Skin," *Brit. Journ. of Dermatology*, September 1891.

reduced, the heart weakened, and the waste of the body increased through all the channels of excretion."[1] According to some it does not reduce the temperature in health. It appears to increase the urea, uric acid, and pigment, while diminishing the water and the chloride of sodium, probably by increasing the perspiration.[2] Ringer, however, says that it is doubtful whether the production of urea is increased, or that its elimination from the body is rendered more free. Observations on this point are not yet conclusive. Its action on chronic skin diseases is thought to be explained by the alterative effects mentioned already.

The direct action of antimony on the skin of frogs was studied by Miss Nunn,[3] and according to her experiments it possessed a deeper penetrative effect than arsenic, which was pretty nearly limited to the columnar cells of the epidermis, while antimony caused softening not only of these but also of those of the intermediate layer.

Such, then, are the few facts which we have been able to glean with respect to this medicine; but a case in which it was employed much in the same fashion, and with results similar to ours, by Dr Kent Spender, needs a passing notice. A lady came under his care at Bath in January 1883, suffering from psoriasis. Her first attack, which had occurred five years previously, had been very successfully treated by the internal administration of tar in capsules, and she had remained quite healthy up to the date mentioned, when she had a fresh seizure, not in any way ameliorated by the same remedy. She was treated by Dr Spender in various ways without any marked change in her condition, till about the end of November, when she suddenly became very much worse, and a state resembling that presented by our first case supervened. On December 1st, one-twelfth of a grain of tartrate of antimony was added to each dose of a mixture containing acetate of potass and colchicum, which she took every six hours. This being well borne, one-sixteenth of a grain was directed to be taken every two hours during the day. The larger dose was commenced on the 3rd December and continued till the 10th without any unpleasant consequences, when the quantity was gradually diminished. On the 7th December, six days after antimony had been first given, a slight improvement already noticed became very marked, and continued without intermission till December 18th, when she left Dr Spender's care practically well. In this instance the progressive advance from psoriasis into general exfoliative dermatitis was described as characteristic.[4]

The most recent observation regarding the action of antimony

[1] Mitchell Bruce, *Manual of Materia Medica and Therapeutics*, 6th ed., 1888, p. 115.

[2] Lauder Brunton, *Pharmacology, Materia Medica, and Therapeutics*, passim.

[3] *Journal of Physiology*, 1878.

[4] *Practitioner*, March 1885.

is one by Mr Malcolm Morris,[1] who expresses the opinion that it is in the cases in which there is a functional nervous cause that antimony is likely to be of use.

Case I.—Psoriasis inveterata, passing into general exfoliative dermatitis, and after the failure of other remedies, completely cured by the administration of antimony.

George D., æt. 17, shale miner. A lad who had always been strong and healthy till his present attack began, and with a family history which exhibited no special peculiarities. He was well grown, well fed, a total abstainer, but perhaps smoked rather much. Except the integumentary, all the other systems were normal. The eruption for which he sought advice at the Skin Department of the Royal Infirmary, on the 3rd October 1891, first attracted his attention about the beginning of September of that year. He stated that it was earliest noticed at the roots of the nails of both hands, though it may have previously existed on the scalp, where, however, it occasioned little or no inconvenience. On the nails and hands it consisted of small red dots, which ran together, becoming white and mealy like on the top. It next affected the arms, which were very itchy, so that he scratched the spots, causing them to bleed. In course of a week it had advanced all over the palms. Within a fortnight the same sequence of events occurred on the face, trunk, and lower limbs. He was admitted through the kindness of Dr Wyllie to Ward 31, and remained there till the 13th October, when he was transferred to the new wards for Skin Diseases. When first seen his condition was as follows:—The hands were swollen, cracked, and bleeding, the palms in particular being covered with masses of thickened, brownish-yellow, horny epidermis. The nails were diseased at their matrices, were claw-like, and worm-eaten at their bases. This rendered him quite helpless, and he presented a truly pitiable appearance. On the arms, the elbows being specially involved, were patches of eruption consisting of brown crusts about a third of an inch thick, seated on an inflamed area, the areola of inflammation extending a quarter of an inch beyond the crust, and gradually fading into the surrounding unaffected skin. On the scalp there were numerous thick, dry crusts, matting the hairs together. On the forehead the condition looked more eczematous, being made up of ill-defined reddish areas, covered with thin brownish or whitish crusts. On the ears there was an eruption of small scattered papules with silvery scales. The margins of the eyelids were reddened, swollen, and crusted, and the commissures of the lips were similarly affected. On the trunk was a copious eruption of psoriasis punctata, while on the legs, and especially below the patella, were several large patches with wide inflammatory areolæ, bearing thick masses of brown crust presenting a laminated arrangement. The soles of the feet were attacked in

[1] *Year-Book of Treatment*, 1892.

the same manner as the palms, the epidermis being enormously thickened, brown, dry, and fissured. The general appearance was, therefore, that known as psoriasis inveterata, characteristically depicted in Tafel 27 of Neumann's *Atlas der Hautkrankheiten*, in which the silvery scales are to a large extent replaced by those in which the keratine has undergone a dark brown transformation.

Under the use of boracic starch poultices the epidermic accumulations became loosened and separated, leaving the subjacent parts smooth, but swollen and vividly red. On the 13th October the diseased portions were directed to be enveloped in rags soaked in a lotion composed of dilute glycerole of subacetate of lead, as recommended by Dr Stephen Mackenzie.[1] This treatment kept the skin soft, but did not prevent the decomposition of the epidermic masses which rapidly re-formed, so that a fœtid odour exhaled from the surface. To counteract this some boracic acid was added to the lotion, and he took warm boracic baths.

On the 19th October the swelling of the hands had slightly diminished, but the skin was still very red, and the diseased condition tended to spread continuously over the face and chest, where there were previously only isolated patches of psoriasis. On the 20th October he had a rise of temperature, apparently due to influenza, then prevalent, checked by acetate of ammonia with salicylic acid. The temperature resumed its normal course on the 23rd. Till the 30th October the boracic baths were continued, when alkaline ones, containing carbonate of potass, were substituted. After these baths he was anointed with purified whale oil, selected for this purpose on account of the penetrating properties which have been ascribed to it. This, though continued for some time, produced no amelioration,—indeed, the dermatitis steadily advanced, and by November 17th had become almost universal. At this date, after the alkaline baths, instead of whale oil an ointment composed of oil of cade, tincture of quillaia, and glycerine of starch was employed. A slight improvement for a time seemed to take place under this treatment, but this was not maintained, and great tension of the skin was produced, so that the eyes could not be entirely closed, or the mouth shut with comfort, and there was difficulty experienced in flexing the limbs. To obviate any danger or inconvenience from the ectropion, boracic starch poultices had to be applied from time to time; these had at least a temporary effect. The skin was at this time extremely itchy, and was scratched to such an extent as to occasion very considerable bleeding. From the 28th November till the 26th December a mixture containing acetate of potass, tincture of nux vomica, and succus scoparii was prescribed, and was regularly taken without any perceptible effect on the skin, or on the secretion of urine, either as regarded its quantity or the amount of urea excreted. On the 9th of December vaseline was substituted for the ointment

[1] *British Journal of Dermatology*, vol. i. p. 298.

of oil of cade, and its employment as an application to the integument after a bath was thenceforward continued throughout.

Up to this point it will be observed that his condition, in spite of all treatment hitherto adopted, had become progressively worse. On the 26th December the skin over the whole body was vividly red, swollen in many parts, here and there tended to crack, and unless kept well anointed with vaseline, the outer layers of the epidermis peeled off incessantly in thin flakes; his state was now that of general exfoliative dermatitis in its most marked and complete form. A new feature, too, had developed, he had become extremely sensitive to cold, shivering when his skin was exposed even for a short time for examination in the usually well-heated ward. This was undoubtedly due to the rapid cooling of the blood in the dilated superficial vessels. He had not previously suffered much from cold before his attack, nor did he in the earlier periods of it. He was at this date placed on tartrate of antimony, one-eighth of a grain being administered every six hours. On the 7th January 1892 the skin of the face had become decidedly paler, and the infiltration was so nearly gone that the eyelids and lips could be easily and entirely closed. The integument of the chest was a pale red, little if any thickening remaining, but the pruritus had not wholly ceased, as there were still evidences of scratching. The same improvement had manifested itself on the legs, the soles and the palms were nearly normal, but the baths and subsequent inunction with vaseline were still employed, as without them there would yet be much flaking off of epidermic scales.

Passing on to the 12th February, a great change for the better could be noted. The head and neck might be said to be normal,—in fact the face was somewhat paler than was natural. The skin of the hands both on the palm and dorsum was healthy in appearance and texture; the nails had resumed their ordinary aspect as regards polish and consistence. On the arms there were still here and there reddish blotches; the chest, back, and abdomen were smooth, yet rather pinker than is customary. On the thighs a reddish mottling predominated, on the dorsum of the feet were some scaly patches, the soles were sound. Latterly the bath and inunction had only been required once in two days, to keep him perfectly comfortable. The itchiness had now quite ceased, he experienced no sense of chilliness on exposure, while his general health, appetite, and capacity for sleep were excellent. On 26th February the skin presented only here and there faintly pinkish macules. These were most pronounced on the shin. The hair was growing thickly, though still slightly scurfy at roots. The scalp was freely movable. On the front of the arm in the bend of the elbow there were a number of very small flat pink papules, slightly but perceptibly elevated, and bearing silvery scales, exactly resembling those seen in a commencing psoriasis of moderate degree of intensity. The antimony was discontinued on March 7th, and

on March 9th he was sent to the Convalescent House as cured, there being then no visible traces of disease.

It should be noted that the improvement consequent on the antimony commenced by a general diminution of redness, but this was not everywhere equally perceptible. Thus on the face—after the scalp, the forehead, and the greater part of the surface were quite of a natural hue—an erythematous blush was conspicuous on the bridge of the nose and malar portions of the cheeks, which faded by degrees till it finally vanished. On the arms also the paler portion seemed to chase away the still persisting islets of redness, infiltration, and scaling, these latter day by day becoming smaller and smaller, till they too ultimately disappeared.

The urea was estimated from time to time by Dr James Mowat and Dr Coleman Moore. It showed considerable variations both before and after the administration of antimony. The only conclusion admissible was, that antimony did not exert any marked influence whatever on the amount excreted; for on several occasions, both before and during the administration of antimony, the quantity excreted rose and fell unaccountably. Nor was the quantity of urine perceptibly affected in his case by antimony.

The temperature was carefully taken morning and evening while in the Infirmary, and was to an appreciable extent modified by antimony. For purposes of comparison the course of the disease may be divided into three periods. First, that during which he suffered from psoriasis only; second, that of exfoliative dermatitis previous to the administration of antimony; third, from the commencement of the exhibition of antimony to the date of his discharge, cured. In the first period, that of psoriasis, the mean temperature was 98°; in the second, the mean was 98°·5; and in the third, 97°·6. From these averages, which are taken from periods of very considerable duration, it is fair to conclude that the dermatitis elevated the mean temperature, while antimony reduced it to or below a normal level, and this apart from any observable functional disturbance whatever. But we have no data from which to determine what was the average normal temperature of George D. in health. During all the period he was under observation he suffered from some degree of dermatitis, for the psoriasis with which he was affected may have raised his temperature slightly, and antimony may have merely reduced it to what to him was natural. Hence our conclusions are not at variance with the opinion of those who hold that antimony does not reduce temperature in health. Only once did the antimony apparently disagree—he felt slightly sick one day, and it was discontinued for twenty-four hours, then resumed, but this may not have had any connexion with the drug.

The result in this case may be considered highly satisfactory when compared with that obtained in similar ones treated differ-

ently. Thus in four cases, two mentioned by Dr Buchanan Baxter,[1] and two recorded by Dr Stephen Mackenzie,[2] of exfoliative dermatitis supervening on psoriasis, one recovered after several months, another got rid of his dermatitis in five months, the psoriasis persisting, a third had improved in a similar period, while the fourth died.

Case II.—Exfoliative dermatitis, tending to spread rapidly, completely cured by the employment of alkaline baths and the exhibition of antimony.

John T., æt. 70, joiner. Admitted to Ward 37 on February 18th, 1892, suffering from a localized exfoliative dermatitis. Family history and previous health excellent. He states that he never perspired freely even when hard at work. The disease commenced three months previous to his admission to the Infirmary, by the formation on the right leg of what he termed "blisters," followed by an itchiness and swelling of both knees, accompanied by a dry, scaly condition of the skin of these parts. Shortly after the commencement of the attack he improved considerably, and this improvement was maintained without intermission till about a month before his admission, when a sudden aggravation occurred. No new blisters were formed, but the swelling, redness, and scaliness of the legs became much more marked, the right hand was attacked in a similar manner, then the left, and subsequently both arms. On February 19th, the day after his admission, his state was as follows:—The epidermis of both palms, soles, and heels, and the dorsum of the second and third phalanges of the fingers was very dry and much thickened, especially on the centre of the right sole, where it formed a thick, dry, friable crust, firmly adherent to the subjacent tissue. The integument of the legs was œdematous, pitting on pressure, the surface reddened, cracked here and there, but not oozing, and covered with thin scaly crusts. On the thighs, buttocks, and pubes were numerous branny scales scattered over a pinkish base. On the arms and back of the hands, as far as the termination of the first phalanges, a similar condition prevailed. The rest of the integument was healthy, but was being rapidly encroached upon by the disease. It was curious to observe the very sharp line of demarcation which existed between the more acutely affected areas of skin and the thickened, dry condition existing on the soles, heels, palm, and dorsum of phalanges already alluded to. There were a few excoriations, but itching was not a marked feature.

He was ordered an alkaline bath of short duration daily, followed by inunction with glycerine of starch, and one-eighth of a grain of tartrate of antimony was given thrice a day. Even already, on the next day, February 20th, the arms were normal in appearance as

[1] *British Medical Journal*, July 26th, 1879.
[2] *British Journal of Dermatology*, July 1889.

regarded colour, and the skin smooth though still slightly scaly. The roughness on the back of the hands was nearly gone. The legs in like manner were also much smoother, the œdema had disappeared, and though the hue was yet somewhat redder than natural, and there was a degree of scaliness present, they had improved almost beyond expectation. This improvement was fully maintained; he was quite well on March 2nd, when the antimony was discontinued, and he was discharged on March 5th. The abrupt line of separation between the thickened and scaly epidermis which had previously been so strongly marked was then quite imperceptible. Before and on admission he did not perspire at all; when he left the Infirmary, his skin in most parts presented a normal, and on the legs a slightly increased, moisture.

He reported himself on the 21st March, and was then quite well as regarded his skin, though he complained of some pain after eating, for which lactopeptine and bismuth were prescribed, with regulation of diet.

The temperature was, in this case, on the evening of admission and on three subsequent evenings, a degree above normal, though it fell to the natural level in the morning. On the fourth day after the antimony had been commenced it became normal at night, subnormal in the morning, and this continued for eight days; then something having gone wrong with the steam, and there being no fireplace in the ward, while the external temperature was low, the atmosphere of the ward was for a day extremely cold. He probably was chilled, as his temperature rose a little, and did not quite return to its previous state for three days, one day after the discontinuance of the antimony. With him, improvement began before his temperature fell, but there was no time to make observations as to the course of his normal temperature either before the antimony was commenced or after its discontinuance.

The amount of urea excreted varied within normal limits, apparently irrespective of the antimony, and the quantity of urine oscillated to a still greater extent. No conclusion could be drawn as to either point.

These cases illustrate the effects of antimony when administered for a prolonged and a limited period respectively, and permit certain conclusions to be drawn and some comparisons to be instituted.

1. Antimony lowers temperature in some conditions of the skin associated with hyperæmia and dryness of the surface, to a well-marked extent.

2. So far as our observations go, its influence on tissue waste as estimated from the amount of urea excreted, or on fluid loss by the kidneys, is not, under the circumstances detailed, a noticeable one.

3. It softens the skin, imparting increased succulence to its cells, augments insensible perspiration, improves the nutrition of the

integument, diminishes hyperæmia, and lessens the tendency to premature and excessive epidermic exfoliation.

4. While advantageous in the early congestive stage of acute eczema, it is contra-indicated during the period characterized by oozing—the second stage of Brocq, that of rupture of vesicles—though it may again prove serviceable at a later era,—the fourth stage of Brocq, that of successive desquamations. We have found this borne out by our experience of a case of eczema treated with antimony, at present in the ward.

5. If Mr Morris is right, as he probably is, that it is likely to be of special use in cases where there is a functional nervous cause, it may prove of value in diffuse scleroderma, and possibly in myxœdema.

6. As compared with arsenic, authors are pretty generally agreed that the latter is valueless in conditions of the pityriasis rubra type,—whether by this exfoliative dermatitis in its dry forms alone is meant, or if pityriasis rubra pilaris is included.

7. Arsenic restrains the tendency to form bullæ in dermatitis herpetiformis and pemphigus, and sometimes cures psoriasis if stationary or a first attack; but, on the other hand, it may apparently sometimes convert a psoriasis into a pityriasis rubra.[1]

8. Arsenic in some cases renders the skin muddy, dull, and earthy,[2] or deeply pigmented; it may induce the formation of horny warts on the fingers,[3] or thicken the epidermis of the palms, giving rise to a keratosis,[4] which again may pass on to epithelioma.[5] Such results have not so far been found to follow the administration of antimony, nor are such likely to accrue.

9. The action of antimony may be contrasted with that of pilocarpine. Pilocarpine produces a copious perspiration for a brief portion of the twenty-four hours; antimony bathes the epidermic cells continuously in a gentle moisture. Pilocarpine lessens or cures a pruritus in a dry, atrophic, anæmic, senile skin, by flushing the emunctories, but its rapid stimulant effect is not suited for cases of active hyperæmia, which, as has been seen, are more amenable to the influence of antimony. Both, however, improve nutrition, and aid in the deposition or restoration of diminished subcutaneous adipose tissue.

Dr Allan Jamieson observed that he had little to add to what had been said in the paper just read by Dr Home Douglas. There were at present in his wards two patients who were being treated very successfully by tartrate of antimony. One was an elderly woman with widely spread psoriasis. In her case the skin had

[1] Hutchinson, *On Certain Rare Diseases of the Skin*, p. 260.
[2] Hutchinson, *Archives of Surgery*, vol. i. p. 76.
[3] *Ibid.*, Plates xviii., xix.
[4] Pringle, *Brit. Journ. of Dermatology*, vol. iii. p. 392.
[5] *Journ. of Cutan. and Ven. Dis.*, 1888, p. 344.

become softer, and the patches of psoriasis had steadily faded under the combined administration of antimony and the employment of alkaline baths of short duration, followed by inunction with various ointments; that which on the whole had suited best was the Unguentum vaselini plumbicum of Kaposi. The other was an instance of general exfoliative dermatitis, which had developed out of a psoriasis of very moderate intensity and considerable duration, in a woman of 30. Her case bore the closest resemblance to that of G. D. in all essential particulars, and like his was rapidly disappearing under antimony. He did not for a moment wish it to be inferred that he proposed antimony as a panacea, but he thought that by it cures could be effected of diseases hitherto regarded as extremely intractable.

Dr Gillespie said that an examination of the blood in all these cases was of great value; and if the hæmoglobin and corpuscles were above normal, depleting remedies to lower the blood-pressure always aided the use of any special remedies. To treat the skin as a special organ was wrong, to treat the state of the blood which nourished it was rather to be aimed at. He had seen several such cases cured by regular doses of purgatives and lowering diet, combined with the then fashionable local remedy; while other cases with a blood below normal in corpuscles or pigment were soon benefited by the ordinary remedies *plus* a stimulating diet and tonics. Antimony probably acted by lowering the blood-pressure, as well as by helping the nutrition of the skin.

Dr Church referred to the chart in connexion with one of the cases of psoriasis, and asked Dr Jamieson if the small secretion of urine, and the abnormally small elimination of urea, as shown by the daily estimation throughout the treatment of the case, had not been a cause of anxiety. Had this diminution to do with the administration of antimony? And if it had, was there not the risk, while successfully treating the skin disease, of running into other dangers by suppressing the normal physiological action of the kidneys? Dr Church joined with the other members of the Society in thanks to Dr Jamieson for his elaborate paper.

Dr Hamilton Wylie expressed pleasure at hearing the excellent results of the administration of tartrate of antimony, and homologated Dr Allan Jamieson's sentiment, that in it we have an additional valuable remedy in treating acute inflammatory diseases of the skin. He thought that the potassium present in tartrate of antimony might have something to do with the valuable results, as in the administration of sulphide of antimony the same effects are not alleged to be produced. The sulphide is usually made use of by veterinary practitioners. Dr Wylie thought it well that Dr Jamieson made a rider, that tartrate of antimony did not agree with every one, and that care should be exercised in its administration.

Dr Douglas replied.

3. NOTE ON TWO CASES OF CEREBRAL HÆMORRHAGE.

By Francis D. Boyd, M.B., M.R.C.P. Ed., Clinical Assistant, Royal Infirmary, Edinburgh.

I venture to bring the two following cases before the Society, as they seem to me to show some points of interest and peculiarity —the first, in the co-existence of a large hæmorrhage into the substance of the brain on left side, with a subpial hæmorrhage on the right side; and the second, in the co-existence of cerebral hæmorrhage and meningitis.

Case I.—J. F., age 65; admitted to Ward VI. of the Royal Infirmary from the eye wards. His wife stated that six days before admission he complained of not feeling well. He said he felt heavy and dizzy in the head. He had been previous to this in perfect health, save for the condition of the eyes, for which he was under treatment at the eye wards. He was at that time quite coherent in his speech. He had always been a temperate man, and, with the exception of rheumatism, had always enjoyed good health. He was taken into the eye wards to have a small operation performed on the eye; but during the first night there he became very restless, getting out of bed. On this account he was sent to Ward VI.

Notes on Admission. — Patient is an old man with arcus senilis well marked. Superficial veins of the face are dilated and prominent. Temporal arteries visible and tortuous. He lies on his back with his mouth half open, and a vacant expression on his face. He seems to understand questions, but answers quite incoherently. Temp., 100°·6. Respiratory system shows slight emphysema; otherwise normal. Sounds of heart are very weak; no murmurs. Pulse 90, very weak; no marked arterial degeneration. Sensibility seems normal on both sides of body. There is slight loss of motor power in the right arm and leg. Plantar reflexes are exaggerated on both sides. Patellar tendon reflex slightly more marked on right side than on left.

On *April 28th* patient had become quite incoherent, and did not seem to understand when spoken to. There was then marked loss of sensation in the right side of the face as compared with the left. He was too comatose to mind prick of a pin on other parts of body. There was now slight rigidity and contracture in the right arm; arm was flexed at the elbow, etc., but this could be overcome with slight amount of force.

April 29.—Unconsciousness has deepened. Pulse 120, very weak; temperature up to 105°. Contracture and rigidity more marked in right arm; present also in left. Still responds to prick of pin about face.

10.30 p.m.—Coma deepened. Contracture and rigidity quite

gone from right arm; it is now completely paralyzed. Contracture and rigidity more marked in left arm.

Patient died on the 30th, temperature before death going up to 107°. He developed no new symptoms.

Post-mortem.—The following condition was found:—The pia was separated from the brain over three-fourths of vertex, and contained excess of cerebro-spinal fluid. There were senile atrophic points in the pia over the vertex. There was blood under the pia on the right side, which extended over the parietal and occipital lobes, and was diffuse. On the left side there was a small subpial hæmorrhage, dividing the parietal from the occipital lobes.

On section a large hæmorrhage into the left side of the brain was found. It occupied a point just posterior to the upper part of the lateral ventricle, and a small rupture in the ventricular wall had allowed blood to pass into the ventricle; but it is doubtful if much blood had passed into the ventricle during life. The posterior half of the internal capsule was pressed upon. The clot in the brain was firm and hard. The vessels at the base of the brain were dilated and atheromatous.

Case II.—D. M'D., age 34; single; a miner. Admitted to Ward VI. November 24, 1891, complaining of "severe pain in the head," which had commenced on the morning of November 23. From that time his mental condition had been peculiar. He said he was getting blind, and seemed to be "off his head."

On Admission.—He complained of severe frontal headache. Was sick, and vomited after admission. Very restless; in a low muttering delirium. Could be roused, and then answered questions coherently. Pupils normal, and react to light. Skin reflexes exaggerated. Patellar tendon reflex well marked. No paralysis anywhere. No physical signs of disease in chest or abdomen. Bowels constipated. No splenic enlargement. Temperature normal. Pulse 72, of good quality. Urine normal. Well-marked sores on penis. Ordered large doses of iodide with bromide of potash; ice to head; purgative.

November 26.—Pupils are now enlarged, and do not react to light. Is very restless, and not so easily roused; does not complain so much about his head. Pulse 64; temperature in the evening, 101°.

November 27.—Restless as yesterday, but to-day the pupils react to light. Blistered behind the ears.

November 28.—Temperature down to-day to 99°·6. Seems more unconscious. Pulse up to 108 per minute.

November 30.—Temperature keeping about 100°, but pulse has gone steadily up. Is more comatose, and cannot be roused. Pulse 140, small and weak.

Dr George Mackay kindly examined the eyes, and gave me the following report:—"Pupils are dilated, and do not react to light.

Discs are compressed and retinal veins full, but there is no actual papilitis."

During the day the patient remained much in the same condition. He was evidently dying, but what was the exact intracranial condition, and could one do anything more with a view to saving his life?

As regards diagnosis, it seemed to me that the patient was suffering from compression, which was progressive, and that the compression was basal. The constipation; the condition of the pulse, abnormally slow at first, gradually increasing in rapidity without corresponding increase of the temperature; the condition of the pupils, dilated and insensitive to light, the optic discs showing congestion, the advancing coma, all seemed to point to this. But to what was the compression due? Was it hæmorrhage? The absence of paralysis, the condition of the temperature, the co-existence of headache with delirium, the early optic neuritis, the vomiting in the early stage of the affection, and the age of the patient, though none of these taken singly would negative cerebral hæmorrhage, still the combination seemed to point more to a basal meningitis with effusion.

This being the case, the clear indication seemed to be to open the cranial cavity, and, if possible, relieve the compression. Mr Caird kindly saw the patient in the evening, and trephined over the left temporo-sphenoidal region. On opening the dura mater about ʒiv. of clear serum welled up into the wound and overflowed. After the operation the pulse fell from 140 to 104 per minute, and became fuller and softer.

On the following day the patient seemed slightly improved. He had never shown consciousness, however, though the pupils reacted to light. He was swallowing better. Ordered stimulants, etc. Towards the evening, however, the condition grew worse, and he began to show symptoms of congestion and œdema of the lungs, with heart failure. He died on the night of December 2, forty-eight hours after the operation.

Post-mortem examination disclosed the following condition:— Brain weighed 3 lbs. 4 oz.; there was excess of fluid at the base. There was meningitis present at the base, as shown by a gelatinoid thickening of the membranes. The vessels at base were normal; no tubercle visible.

There was a large hæmorrhage into the occipital lobe on the right side, which had burst into the right ventricle, and seemed to be of some days' standing. No recent hæmorrhage. Microscopic examination of the vessels disclosed no abnormality.

The occurrence of two independent hæmorrhages into the brain, as in Case I., is a comparatively rare condition. We have here a very good clinical example of the effects of pressure on the internal capsule: first, paresis, then rigidity, and as the pressure gradually increased, complete paralysis. On the opposite side we have the

effects of a small superficial lesion, giving rise to symptoms of irritation, as shown by the contracture on the left side, which came on thirty-six hours after the rigidity on the right side.

The second case seems, as far as I can discover, to be an almost unique one, as I can find no records of a similar co-existence of meningitis with cerebral hæmorrhage, the two being apparently independent of one another.

The history seems to point to the hæmorrhage having taken place on the 23rd. He then complained of getting blind. Had he been examined, homonymous hemianopsia would probably have been discovered. Examination of the vessels, both naked-eye and microscopic, gave a negative result, though the fact that the patient had syphilis would lead one to think that there had probably been some syphilitic arterial degeneration, and that the vessel had given way at the weakest point.

What was the cause of the meningitis? No tubercle could be found, and no evidence of tuberculosis in the other organs. Some observers hold that all non-traumatic meningitis is due to tubercle, but my experience does not bear this out. During the epidemic of influenza we had in Ward VI. a case of basal meningitis, which undoubtedly came on during an attack of influenza; and at the post-mortem no evidence of tuberculosis could be found either in the brain or in the other organs.

The theory of this case which I would venture to suggest is that, in the first place, there was meningitis, possibly due to influenza, with accompanying cerebral congestion; and that during this congestion the vessel within the brain gave way at a point weakened by disease, the disease being possibly a syphilitic periarteritis. A point which I think is worthy of note in this case is, that though the intracranial pressure was suddenly relieved, there was no fresh hæmorrhage. My only regret is that the patient was not trephined earlier. He died of cardiac failure and œdema of the lungs. If he had had a better constitution to work upon, he might have been another feather in the cap of cranial surgery, and, instead of dying, been living, cured of his disease.

The President remarked on the excellent, brief, and yet precise manner in which the cases were detailed, and expressed his opinion as to the importance for the study of cerebral localization to have records like this of cases watched by competent observers.

Dr Russell said that the careful and accurate recording of cases of cerebral lesion was of great importance and value, and he hoped Dr Boyd would continue to record the interesting cases he might see from time to time. Such records did not provoke discussion, but the more one saw of cerebral cases in the *post-mortem* room, the more cautious it tended to make one in forming an absolutely certain diagnosis.

Meeting IX.—May 4, 1892.

Dr Clouston, *Vice-President, in the Chair.*

I. Election of Member.

Dr G. Matheson Cullen, 48 Lauriston Place, was elected an Ordinary Member of the Society.

II. Exhibition of Patient.

Mr Caird showed a patient, aged 29, who was admitted to Hospital six days after he had sustained a simple transverse fracture of the olecranon by a fall from a height of 18 feet. The patient's circumstances required that he should be able to return to his work as soon as possible, and he desired to have the fracture wired. It was therefore cut down upon, and the separated fragment was found to be split longitudinally. The portions were wired together and the wound closed. Patient left Hospital in a week, and on the fourteenth day after the operation he was able to execute a full day's work, to handle the saw and follow out his occupation, that of a joiner, with perfect use of the joint.

III. Original Communications.

1. REMARKS ON THE SURGICAL TREATMENT OF GENERAL PARALYSIS OF THE INSANE.

By John Macpherson, M.D., F.R.C.P. Edin., Stirling District Asylum, Larbert, and David Wallace, F.R.C.S. Edin.

In submitting the following record of our experience in the surgical treatment of general paralysis in the Stirling Asylum, we consider it proper to state our reasons for the performance of these operations. After carefully considering the subject from different aspects, we concluded that some further acquaintance with the operation and its results was desirable. The pathological conditions usually found and generally believed to exist in the disease appeared to give countenance to the supposition that surgical interference might interrupt or permanently relieve the affection. The operation had been shown by the previous experience of others to be unattended with any danger, provided, of course, that it were carefully performed, and that ordinary attention was afterwards given to dressings and nursing.

Briefly stated, we considered that the operation was justified—(1), because the pathology of the disease seemed to mark it as capable of relief by surgical operation; (2), because further acquaintance with the results of operative interference was desirable in the present state of our knowledge; (3), because of the proved safety of operative treatment.

General paralysis is a disease of the brain cortex, chiefly affect-

ing the fronto-parietal regions. The disease in most cases is limited to areas on the vertex of those regions. Whether it is regarded as of nervous origin, or looked upon as a primary inflammatory affection of the non-nervous elements of the cortex, the limitation of affection is capable of explanation on the generally accepted theory, of which Meynert[1] is the chief expounder—that certain portions of the cortex may be the seat of functional hyperæmia, while other (even neighbouring) areas remain non-hyperæmic. It is more than likely that these portions of the cortex in front of the fissure of Rolando are chiefly concerned in what we call, for want of a better name, the higher mental processes. The experiments of Goltz and of Ferrier seem to bear this out and to strengthen the view. We may, in addition, cite the weighty authority of Ross.[2] Further, it is believed by many psychologists that the motor areas of the cortex underlie, to a great extent, the higher mental processes. If, now, we consider general paralysis in the light of a mental, or, more properly, a mental motor affection of the brain cortex, we come *a posteriori* to a plausible explanation of why that particular region is more chiefly affected.

Whether the disease is of primary nervous origin or not does not appear to be of very essential importance, except in so far as it directly bears upon the results expected from surgical interference. Some authorities hold that the disease is a degeneration of the nerve elements of the cortex, and that all the phenomena are explicable and ought to be assigned to that cause. The chief modern upholders of that view would appear to be Drs Clouston and Savage.[3] Other authorities again hold that the affection is primarily a vascular one—that is, inflammatory, and that the nerve cells are secondarily affected. We believe we are justified in placing Dr Bevan Lewis[4] among the upholders of this theory. We have been unable to decide to which view Mickle[5] inclines, as his judicial summing up of authorities on this point is a little confusing. The following quotation, however, epitomises his opinion,—"On the whole, we may view general paralysis as essentially commencing with hyperæmia and ending with chronic cortical degenerative cerebritis."

Granting that the disease is primarily nervous, the result of morbid over-activity of the cortical cells would be hyperæmia of special localities, for, as Meynert[6] points out, not only is the anterior cortex a vaso-motor centre, but each region of the cortex controls its own blood-supply, so that when an associated area is active, it is at the same time hyperæmic. If, however, we adopt the theory of primary vascular affection, we see that, as a result

[1] *Psychiatry*, p. 214.
[2] *Diseases of the Nervous System*, vol. ii. p. 346.
[3] *Journal of Mental Science*, vol. xxxvii. p. 495.
[4] *Textbook*, p. 439.
[5] *General Paralysis*, p. 342.
[6] *Psychiatry*, p. 216.

of this congestion, the nerve cells, owing to their unique method of blood-supply, through the perivascular canals, must suffer (as they actually do) and be thrown into a state of morbid over-activity, which ought, according to the advocates of the nervous theory, to augment the existing state of congestion of the blood-vessels. We know, on the authority of Duret,[1] that in certain cases of cerebral concussion the resulting vascular paralysis may pass on to inflammatory congestion. Therefore, whichever view of the origin of this disease is entertained, it must be acknowledged that there is vascular inflammatory congestion. It must be admitted that if the origin of the disease is a primary nervous degeneration followed secondarily by inflammation, surgical treatment would not be so hopeful, for, while relieving symptoms, it could hardly be expected to do more than check the inherent degeneration of protoplasmic vitality. It seems probable, however, that there is something more than passive inflammatory congestion of the bloodvessels of the affected region to be considered. The condition presents a more active pathological aspect than can be accounted for by mere passive congestion, and points to the presence of an inflammatory stimulant.

Dr Bevan Lewis says that the earliest indication of morbid change is certainly presented by the vascular tissues. There is, first of all, a true paralysis of the tunica muscularis of the cortical arteries (pial vessels) accompanied by extensive protoplasmic segmentation and nuclear proliferation. There follows, as a consequence, a transfusion of the fluid contents of the vessels into the perivascular canals, and from thence into the pia-arachnoid spaces into which these canals open. Finally, there is a diapedesis of leucocytes into the perivascular canals and the above-mentioned spaces.

Is this, then, a true primary active inflammatory condition of the vascular tissues, or a passive congestion, the result of a vaso-motor paralysis due to primary affection of the cortical cells which have undoubtedly a vaso-motor function? Our opinion is that if such a vaso-motor paralysis could be admitted to account for the inflammatory state, death would ensue more speedily than it does from increasing paralysis.

The cause of such an inflammation is as yet unknown, but, according to the latest researches, there is probably always present in inflammation an organism which produces or accompanies the characteristic phenomena which we recognise as inflammatory, and we must not consider the brain tissue so sacred as to be immune from influences which affect other regions of the body.

We are, therefore, inclined to think that the changes are due to a specific inflammation, chronic in its course, which seizes upon the vascular tissues of regions weakened, it may be, from lack of proper innervation as well as by too frequent hyperæmia due to excessive or too frequent functionating, and we conclude that in

[1] *Traumatismes Cérebraux*, p. 56.

the earlier stages of general paralysis we have to deal with an inflammation which chiefly affects the bloodvessels of the fronto-parietal region.

Two pathological consequences, both highly detrimental to the brain cortex, result from this inflammatory affection of blood-vessels. First, there is an interference with the nutrition of nerve cells; and, secondly, there is an effusion of fluid which causes injurious pressure upon the brain within the cranial cavity.

The symptoms of general paralysis, in its early stages at any rate, depend upon the arrest of the free lymph circulation in the perivascular canals and the pericellular sacs. This interference with free circulation is due—(1), to the dilatation of the paralysed arteries in the canals; (2), to the inflammatory accumulation of leucocytes in the canals; and (3), to the nuclear proliferation of the cells of the tunica adventitia of the vessels.

Later on, in the course of the disease after the degeneration of the nerve cells has become pronounced, the growth of spider cells interferes still further with the lumen of the canals, and produces kinks in the course of the arteries; but with such an advanced stage we are not presently concerned, for by then operative measures would cease to be of any presumable benefit upon any recognisable hypothesis except that of the restoration of degenerated cortical tissue, which is in the present state of our knowledge impossible.

It must be remembered that not only is the supply of fresh nutritive material to the cells diminished in the manner described, but that the removal of the waste products of protoplasmic metabolism is also seriously diminished.

The cerebral lymph is normally an alkaline liquid, but becomes acid after death,[1] and it would appear that in certain pathological conditions the same change occurs.[2] Bevan Lewis[3] states that the apparent cell degeneration is secondary to the affection of the vascular tissues. There can be no question that malnutrition and poisoning by its own waste products must exercise upon the nerve cell an effect which ends in speedy degeneration.

Former writers on this subject, notably Dr Batty Tuke and Dr Claye Shaw, have regarded the removal of the accumulated fluid the chief object of operative interference in general paralysis. While far from ignoring the importance of the presence of the fluid, we do not give it the first place as a baneful pathological factor. There are two stages of fluid exudation, namely, (1) the inflammatory exudate, and (2) the compensatory fluid exuded to replace shrinkage of cortical tissue caused by nerve cell degeneration.

These two fluids are as yet indistinguishable from one another,

[1] *Vide* Meynert, *Psychiatry*, p. 235.

[2] Heynsius and Preyer quoted from Dr B. Tuke's article, *British Medical Journal*, vol. i, 1890, p. 10.

[3] *Loc. cit.*, p. 493.

but we do not agree with Dr Claye Shaw when he admits (in the description of one of his cases) that the fluid must be compensatory.[1] If the fluid was compensatory it was too late to perform any operation, as the disease had advanced beyond hope of human restoration. Mr Harrison Cripps[2] commits the same mistake when he asserts that the brain substance is diminished in general paralysis, and suggests that the atrophy is caused by the pressure of the fluid.

In order to form a comprehensive and intelligible view of the pathology of the disease, especially as it bears upon the question of surgical treatment, we think a careful discrimination must be made between the inflammatory exudate and the undoubted compensatory fluid that is continuous with, but comes on subsequent to the inflammatory exudate, and is consequent upon brain shrinkage. With regard to the effects of pressure from the accumulation of fluid within the cranium we are unable to form any definite opinion. According to Leyden,[3] the tension of the cerebro-spinal fluid is equal to a column of water 10 to 11 centimetres high. Dr Batty Tuke[4] makes a calculation by which he endeavours to show that were the intracranial pressure equivalent to 100 atmospheres, the withdrawal of the $\frac{1}{220}$th of its bulk would reduce its pressure to that of the atmosphere. Without attempting to criticise this statement in any way, we would point out that there is within the cranium a self-regulating mechanism by means of which, within certain limits, fluid pressure can be regulated. The tension of the cerebro-spinal fluid depends upon the blood pressure, and can be reduced by bleeding.[5] But according to Duret,[6] pressure upon the surface of the brain increases arterial tension very markedly. The same author states[7] that at a pressure of 15 centimetres of mercury, half a litre of water injected into the arachnoid space of a dog was speedily absorbed, proving the rapidity of the absorption of fluids by the serous cavity of the arachnoid. In another part of his work the same author,[8] illustrating the great adaptability of the brain to pressure, shows that the injection of 120 to 130 grammes of blood into the arachnoid cavity of a dog produced no symptoms, while 240 to 250 grammes of the same fluid caused coma, followed in a few hours by death. We therefore conclude that in general paralysis the production of the fluid must either be enormously increased, or its rate of absorption by the extremely absorbent pia-arachnoid sac must be in some way retarded, to render the inflammatory exudate injurious by its pressure. At present we have no means of ascertaining either the rate of increase or the rate of absorption of such fluid, but we know that it is abnormally produced, and

[1] *British Medical Journal*, vol. ii., 1891, p. 581.
[2] *Ibid*, vol. ii., 1889, p. 1215. [3] Virchow's *Arch.*, 1866, Bd. 37, p. 520.
[4] *British Medical Journal*, 1890, vol. i. p. 9.
[5] Duret, *Traumatismes Cérébraux*, p. 23; also Dean, *Path. Journ.*, vol. i. p. 35. [6] *Loc. cit.*, p. 166. [7] *Loc. cit.*, p. 175. [8] *Loc. cit.*, p. 202.

that its presence is probably injurious, not only by exercising pressure, but also by waterlogging the vital processes of the cortex.

Up to this point we have regarded the brain and the contents of the cranium as an immovable mass within a closed bony cavity. Within such rigid walls no increase of fluid would be possible were it not for the basal lymph cisterns, which permit a certain amount of ebb and flow of their contents by adapting themselves to an increase or decrease of pressure. Further, there have to be considered in this connexion the venous spaces of Cruveilhier adjoining the sinus longitudinalis. These cavernous spaces are regarded as a compensatory mechanism designed to secure at all times the repletion of the cranial cavity. We must also take into consideration the rhythmic movements of the brain, which are three in number: 1. The systole and diastole, a tracing of which can be got as accurately from the surface of the normal brain as from the radial artery, showing the tensity of the cortex. 2. The respiratory rise and fall, a very distinct movement, 15 to 18 per minute. 3. The vascular wave, a peristaltic rhythmical movement dependent upon a subcortical vaso-motor centre, and passing over the whole brain from two to six times per minute.

We must therefore consider the living brain as a semifluid body subject to considerable expansile movements of frequent occurrence, contained within a bony cavity which permits only a limited change of capacity. It is evident that a very small opening in the cranial chamber will greatly relieve the tension of a moving expansile body subject to such pressure.

From the fact that the convolutions of the vertex impress their mark upon the inner table of the cranial vault, while the basal convolutions do not do so upon the base of the skull, we infer that the interposition of superfluous fluid between the vertex of the brain and the cranial vault must inflict injurious pressure upon the former. If, then, there exists in this disease a true chronic vascular inflammation of cortical areas, causing an interference with nerve-cell nutrition, and exudation of an abnormal fluid causing pressure, it seems a clear duty to attempt to relieve the inflammation, which is admittedly the origin of the whole chain of fatal symptoms met with.

The admitted failure of therapeutic measures justifies a trial of those surgical methods commonly practised for the relief of inflammatory conditions in other parts of the body, namely, the artificial relief of tension. As already stated, we desire to discriminate between relief of the inflammatory condition and the relief of injurious pressure by the removal of superfluous fluid. It has been stated as an objection to this method of treatment that the comparatively small quantity of fluid removed was insufficient to relieve pressure, and would speedily be replaced by re-accumulation of fresh fluid. In reply to this objection, we can

only affirm that we were guided by the analogous treatment of apparently similar conditions in glaucoma and acute effusions into the pleural cavities.

Dr Percy Smith,[1] in criticising the results of Dr Claye Shaw's and Mr Harrison Cripp's operation, emphasised by the aid of a series of illustrative cases the frequency of the total remission of all the symptoms in the course of general paralysis. In our opinion, this formed one of the strongest arguments in favour of the performance of the operations, for the very fact of the possibility of the remission of the mental and motor symptoms is fatal to the primary nervous degenerative theory of the disease, and held out a prospect of possible amelioration by the relief of the inflammatory condition. Finally, in the words of Professor Ferrier,[2] the disease is so fatal that any experimental attempt to relieve it is justifiable.

In all five patients have been treated. We shall first describe one case typical of all, and then point out a few of the chief features regarding each patient.

Case II.—P. McL., male, aged 32, married, was admitted into the Stirling District Asylum on September 22nd, 1891. Two years previous to admission he sustained a blow on the head by a bale of goods falling upon him. His friends date his insanity from the time of injury. There is no scar nor sign of this injury to be discovered. Deep and superficial reflexes exaggerated; speech tremulous, but patient able to repeat test words as ordinarily used with comparative ease; smell impaired to a considerable extent, but taste is only slightly deficient; common sensibility is increased. Mentally he is weak and facile; he has many delusions of grandeur, and is placid and contented; he steals useless articles in a clumsy, silly manner; he is often noisy, and is constantly buttoning and unbuttoning his clothes; says that he is "up to the knocker," etc. Slight evening rise in temperature (98°·8 to 99° F.) generally recorded.

On *October 9th*, 1891, the patient's head was shaved and thoroughly washed with soap and water, and then with ether, after which a carbolic poultice (1 to 20) was applied for twenty-four hours.

On *October 11th*, half an hour before the operation, $\frac{1}{4}$ grain of morphine was given subcutaneously. Anæsthesia was produced by chloroform, which the patient took without trouble and stood well. By Hare's method the lower end of the fissure of Rolando was mapped out on both sides of the head, and an extensive semilunar flap, with the convexity upwards, was made on each side to expose the corresponding portions of the skull. The scalp was reflected without turning back the periosteum. Over the orolingual centres—inferior part of ascending frontal and posterior

[1] *British Medical Journal*, vol. i., 1890, p. 11.
[2] Address at the University of Edinburgh, 1892.

part of left inferior frontal convolutions—a portion of periosteum 2 × 1 inches was removed, and then with the trephine, gouge, and gouge forceps a portion of the skull cap of corresponding size was taken away. Bleeding from the soft parts was arrested temporarily by forcipressure. The dura mater on exposure did not bulge more than normally into the opening, but looked whiter and denser than usual. From the corresponding place on the right side a portion of skull was now removed by the same method 1½ inch square.

The exposed dura was similar in appearance to that on the left. The bleeding from the diploë was completely arrested by Horsley's putty, and then the dura on the left side was opened up by a crucial incision. At the moment of puncture of the dura, a quantity of clear limpid fluid escaped. At first it spouted out for an inch or more, and then gradually flowed over the wound. The pia arachnoid was vascular in appearance, and more thickly supplied with bloodvessels than usual; along the veins there was milkiness. When the pia was incised a small quantity of fluid escaped from the sulci; over the summits of the convolutions the pia was adherent. The cerebral convolutions were not atrophied.

A similar proceeding to the above was now carried out on the right side, with precisely the same results, but the fluid which escaped was considerably less.

The dura on each side was now cut away to an extent which merely left its margin projecting from under the cut surface of bone. The skin flaps were stitched into position, gentle pressure applied by means of the dressing, and the patient was returned to bed.

After-Progress.—The patient's temperature, which had been irregular previous to the operation, rose to 99°·8 on the right side, and to 100°·6 on the left by 8 P.M. (that is eight hours after the operation). In the morning at 8 A.M. on the right side the thermometer registered 99°·4, and on the left 99°. From then onwards the temperature for five days remained a little above 98°·6 in the morning and 99°·2 in the evening. It was taken every four hours for a week. The patient complained of no pain, and the dressings remained in position, therefore the wounds were not dressed until the eighth day from the operation, when they were found to be perfectly healed from end to end. During these eight days there was no improvement in the mental or motor symptoms.

Present State (May 1892).—Reflexes extremely exaggerated, right pupil wider than left. The tongue is projected spasmodically. Speech is very tremulous. Pupils react to light and accommodation. Movements slow and deliberate. Mentally he is demented, his power of attention and curiosity is entirely lost. He is stupid, and his memory is much impaired. Notwithstanding this, an amelioration took place in all his symptoms about six weeks ago, when he became much less restless, and his habits, formerly dirty, are now clean.

Surgically, the case which has been recorded differs only in

small particulars from the other four of the series. In Cases I., II., and III. one side of the head only was opened, the left, but the procedure in all of these was precisely similar to that which has just been described.

Case V.—In this case both sides of the skull were opened and portions of bone removed on the left side 2 inches by 1½ inch, on the right a circular portion 1½ inch in diameter. The dura, however, was not removed, only freely incised, and a horsehair drain introduced on each side between the dura and pia arachnoid membranes, resting on the pia arachnoid in the form of a loop, and brought out at the most dependent parts of the wounds. Dr Batty Tuke, who was present at this operation, made the following notes:—"Right side: bulging of dura not more than normal; on palpating dura finger readily detected layer of fluid, and on puncturing, flow of fluid readily produced; on opening dura on right side two layers abnormally distinct; brain pulsation visible; arterial and venous congestion under pia arachnoid membrane, which bulged on removal of dura; on puncturing pia, fluid escaped and brain pulsation greatly increased; arachnoid pia not milky. Left side: Respiratory change in brain pulsation much more distinct than before the right side was opened; on opening dura the arachnoid pia was found more milky and showing much more intensely indications of inflammation; pinkness; injection of vessels. Amount of fluid very small; apparently drain had been made from the other side." In this case the head was dressed on the third day to see whether drainage was satisfactory, but by the eighth day the wounds were firmly united.

There are some points in connexion with the operation which seem worthy of note. In the first place the site selected for operation was chosen because, first, almost invariably in general paralysis the tongue and lips suffer from motor symptoms before the other muscles of the body; and, secondly, because relief of pressure by the removal of fluid could be carried out at that part as satisfactorily as elsewhere. So far as the operation itself is concerned there seem three points worthy of attention:

First, the large semilunar flap gave great support to the portion of the brain exposed, and although necessarily a large surface of periosteum was exposed, the cut in it and the reflection of a portion was not greater than the size of the piece of bone removed.

Secondly, large portions of the cranium can be removed very rapidly by the method adopted—namely, a combination of trephine, gouge, and gouge forceps. First of all, a circle of bone is removed by means of the trephine. This can be done more rapidly than usually is the case, because the trephine circle can be levered out before the bone is completely divided, and if any splintering of the internal table do occur, the sharp spicules are removed with those parts which are taken away by the gouge. The circle of bone

having been cut away, the thickness of skull with which the surgeon has to deal is known, and with the gouge and hammer very quickly the bone to any extent can be cut away. Preferably by this means only the outer table and a portion of the diploë are removed, the remaining diploë and inner table being then snipped off with the gouge forceps.

The gouge forceps used were a modification of those in common use, the inferior blade being flat on the surface and not curved; this has the advantage of allowing the blade to be more easily introduced between the dura and the inner table than is the case with the ordinary forceps. Hoffman's forceps, perhaps, have even greater advantages.

Thirdly, the bleeding from the diploë, except that from very large veins, was readily arrested by means of a putty of the following composition: white wax ʒij., yellow wax ʒiv., carbolic acid ʒss., vaseline ℥ss. A note of this composition was found in a paper by Dr Keen of Philadelphia, where he speaks of it as Horsley's putty.

The following seem to be the chief points of interest in these cases apart from the after-result:—

1. The *bone* varied in thickness, density, and vascularity considerably in three cases, but in only the first was there anything specially noteworthy. In it there was complete absence of diploic tissue, and, as a consequence, great density and little vascularity.

2. The *dura mater* in all was adherent to the inner table, but in no case was it extremely so. In all it was dense and thick—in our opinion, denser and thicker than the normal membrane. In no case did bulging into the opening, as described by Drs Batty Tuke and Claye Shaw, occur. We believe, however, that bulging of the dura cannot be expected even though there be increased intracranial pressure, unless the dura at the part be so thin as to be capable of stretching, inasmuch as the dura around the opening is adherent to the inner table, and without separation from it, or stretching of the part exposed, it cannot bulge. Further, as a rule, the dura mater is not only thicker in general paralysis, but it is more adherent to the bone.

3. *Fluid* in all the cases, when the dura was incised, escaped in variable quantity. It was a clear limpid stream of serous fluid, which contained no flakes. In two cases the fluid spouted out for a distance of at least one inch.

In Case V., after the dura was incised on the left side and the subdural fluid allowed to escape, some time was taken to closely examine the pia arachnoid, and to note the pulsations of the brain; and later, when the dura on the right side was cut into, very little fluid escaped, and at the moment of puncture it was observed that the brain surface was not apposed to the under surface of the dura. It would seem probable, from a consideration of this case, that a very free communication between the fluid on the two sides

of the head exists. In Case III., after the dura was incised, great bulging into the trephine opening occurred, so that the appearance of a cyst was produced. This was due to the opening being immediately over a sulcus, and to the fluid under the pia arachnoid membrane forcing that membrane upwards. On opening the cyst-like swelling the fluid which escaped resembled the fluid from the subdural space.

4. The *pia arachnoid* membrane in every case was distinctly redder and more vascular than usual. In two cases there was a milky appearance along the vessels. On incising the membrane, fluid similar to that in the subdural space escaped, but only a small quantity. In two cases where an endeavour was made to separate the pia from the summit of the convolutions, it was found impossible to do so. In regard to the greater congestion of the pia apparent in these cases, it should be remembered that the intra-cranial pressure having been much diminished, it is possible that some dilatation of the vessels occurred, giving rise to a pinkness, very marked in Cases III. and V., which might have disappeared had they been longer under observation, the vessel walls regaining their tonicity.

5. *Pulsation* in every case was less than normal before the dura was opened, but after the fluid was evacuated, pulsation was marked. In Case III. it became so alarmingly influenced by respiration, that it was thought advisable to open the other side of the head. In this case the sucking in of the brain during inspiration produced a depression of about three-quarters of an inch.

From a consideration of the above series of cases we have come to the following conclusions:—

Surgically, all the cases have progressed uninterruptedly to recovery. At the end of eight days the wounds were in every case firmly united, and in none was there a drop of pus. None of the patients seemed to suffer in the least degree from the operation. Undoubtedly, much of the surgical success was due to the able assistance which we from the outset received from Dr Skeen, assistant physician at the Asylum. We are deeply indebted to him for the assiduity with which he carried out all our directions, the assistance he gave at the operations, and the untiring interest he has taken in the after-progress of the cases.

Medically.—In all the cases, with one exception (No. 2), there was a marked improvement in the mental symptoms, lasting for from one to three weeks. This may be fairly accounted for as the result of the operative procedure. We consider that the relief of pressure by the removal of fluid, the greater freedom allowed by the removal of bone for cerebral expansion and pulsation, and, perhaps, the relief of the inflammatory condition by the local depletion of the vessels of the scalp, diploë, and brain membranes, necessitated during the operation, may all have combined to produce the change we noted.

It is significant that concomitant with the cicatrisation and increasing density of the scalp and fibrous membranes over the openings there took place a gradual deterioration in the patients' mental and motor symptoms. In Case V., where an attempt was made to drain the pia arachnoid sac by means of horsehair, the difficulty of preventing rapid healing of the wounds over the horsehair was found to be insuperable, and made its function of draining nugatory. Therefore we are unable to state from our experience whether such a mode of drainage would be beneficial.

We are inclined to regard the removal of subdural fluid a means towards the alleviation of the inflammatory condition of the pial vessels involved, and not the primary object of the operation. In connexion with this we have noted that immediately after the operation, and in the after-history of the cases up to the present date, there has been no bulging of the scalp over the openings, which one would naturally have expected from the over-pressure of fluid; but, on the contrary, that the scalp has been sucked in, so to speak, and formed a cup-shaped depression. Again, as the membranes have hardened over the openings, the pulsation of the brain has become less and less apparent on palpation. The absence of bulging and pulsation might be accounted for by shrinkage of the brain tissue proper were it not that the depression occurred from the first in cases in which there was no apparent atrophy of the convolutions.

That this cup-shaped depression is not due to adhesion of the scalp to the underlying membranes of the brain we are justified in stating, from the result of the *post-mortem* examination of Case I., who died three months after the operation, and in whom no such adhesion was found. The absence of adhesion is, of course, fatal to Mr Harrison Cripps's theory that a lymphatic communication might be established between the membranes of the brain and the scalp tissues.

We regret to record, as the result of our experience, that no permanent or marked benefit was conferred upon our patients by the methods of surgical treatment we were led to adopt in the manner described. In Case III. we believe that the motor symptoms of the disease were, and continue up to this date, relieved by the operation, but the delusional state is as bad as ever. We do not wish to discourage others from further attempts in the same direction. In our hands the operations proved, from a surgical point of view, eminently successful, and no bad results of any kind were suffered by our patients.

We would remark, finally, that it seems as if the operation, to be of material benefit, should be performed at an earlier stage of the disease than in our cases. In all the cases we trephined, the pathological appearances were such as to lead us to infer that the disease was fully established. We believe that the present state of our knowledge of general paralysis, and our power to diagnose

the disease at a sufficiently early stage, is so imperfect that as yet surgical treatment—at all events, by the method adopted by us—can be of no material benefit whatsoever.

Dr Ireland observed that these operations afforded another proof of the safety with which the vault of the skull could be opened under proper precautions. Formerly surgeons were very timid of trephining, and it was the localisation of function of certain areas in the brain which furnished the occasions for surgical interference. The best results were obtained where there was a clear indication of the site of the lesion, as in tumours and abscesses of the brain. In general paralysis they had a disease of the cortex, inflammatory, or at least having a hyperæmic character, which in the end spreads to the whole nervous system. Dr Macpherson, whose pathological paper might be viewed as a justification of the operation, said that the disease began in the frontal lobe, which might be. But from the symptoms it seemed to begin sometimes in one part of the cortex and sometimes in another, and the hemispheres were often unequally affected. Hence there was no clear indication for the surgeon where to open the skull and what he would find. What was proposed was to relieve two symptoms which were not the essential symptoms. To relieve the brain of the pressure of the arachnoid fluid it did not seem necessary to make so large an opening, and though surgeons in despair at the persistence of a hyperæmic condition sometimes tried the effect of a free incision, he doubted whether the brain tissue would stand such treatment. Nevertheless he thought that in so desperate a disease as general paralysis the interesting experiments detailed were quite justifiable. Dr Ireland thought that the operation of opening the cranium might be tried with good results in that rare disease, hypertrophy of the brain.

Mr Chiene said the surgical aspect was perfect and called for no remarks: he left it to the psychologists to discuss the advisability of the procedure.

Dr James stated that although he agreed with the authors of the paper, that theoretically benefit might be expected from trephining in general paralysis, he was inclined to believe that this disease was associated with a diminished rather than an increased pressure in the cranial cavity. This was borne out by the observation that in some of the cases it was found that after the operation, although the symptoms continued, the covering of scalp over the trephine opening showed a depression and not a bulging, as would have occurred had increased pressure been existent. He thought that diminished pressure better agreed with the pathological and clinical phenomena. The disease seemed to be due to a failure in the trophic power of the brain matter, and this must lead to a shrinking in bulk; but even were it due to those little understood changes which we designate by the name of inflammatory, he be-

lieved that a similar diminution in bulk must eventually ensue. The operation of trephining would, however, theoretically be none the less beneficial, because it would permit a gradually atrophying brain to shrink, with as little disturbance as a shrivelling skin permits senile wasting of the trunk and limbs. They must not forget that the hard, unyielding skull has, like everything else in Nature, effects for evil as well as for good. It protects the delicate brain from injury, but it prevents the little alterations in bulk which occur again and again harmlessly in other tissues; it leads to the formation of miliary aneurisms; it favours hæmorrhages and intensifies their evil effects. Indeed, if it be not considered too transcendental, they might even believe that, as civilisation progresses, the evil effects of a hard, unyielding skull will become more and more apparent. With the advance of civilisation, with the diminution in the physical element, the risk of knocks on the head will become less and less, whilst the necessity for rapid alterations in the blood-supply and consequent bulk of the brain as a whole or at different parts will become more and more pressing. They may even suppose that in the distant future the evil effects of this hard, unyielding bony skull may become so prominent that by the physician of that period trephining will be considered as necessary a practice for the welfare of humanity as vaccination is by the physician of the present day. That was, of course, too hypothetical to be scientific; but there was no doubt that a very elementary study of the physics of the cranial cavity only was required to demonstrate that in many pathological conditions of the brain the operation of trephining may be of great use.

Dr George M. Robertson congratulated the authors on their paper, and on the skilful surgical treatment of their cases. Excepting from an experimental point of view, he did not think the operation justified on the grounds mentioned in the paper. The operation was proposed to relieve the pressure on the surface of the brain caused by the cerebro-spinal fluid, and not above one or two ounces—the exact amount was not stated—had been thus removed. It was known from experiment and also clinical experience that the rate of secretion of this fluid was very rapid, hence the good done by this operation could only be very temporary. It was also known, from experiments by Duret and Adamkiewicz, that the rate of absorption was also extremely rapid, hence this operation was uncalled for. Moreover, if a momentary drainage were the only object, why make so large an opening? The fact is that the theory underlying the operation is wrong,—the cerebro-spinal fluid is a passive agent for regulating intracranial pressure, and if there is increased pressure it must be due to an increased amount of blood. This possibly exists in the early stage, and it could be relieved by a large removal of the skull-cap. In the later stages we have convincing proof that there is decreased pressure, and at this stage we have an increase

of cerebro-spinal fluid of a compensatory nature. The most efficient way of removing this is to tap the spinal arachnoid, but he did not believe the true pathology of general paralysis justifies this operation.

Dr James Ritchie said that in chronic hydrocephalus the nerve elements were subjected to a considerable amount of pressure for a lengthened period, but yet the symptoms were widely different from those which were exhibited by a person suffering from general paralysis. He was therefore unable to accept the theory which ascribed the disease or its symptoms to increase of intracranial pressure. He thought that it could not be a primary nerve degeneration, that there must be some prior cause inducing congestion. If it were purely a nerve degeneration there would not be the periods of quiescence, with, in some cases, apparent recovery.

Dr James Middlemass questioned the existence of increased intracranial pressure in general paralysis, and quoted recent experiments of Adamkiewicz in this direction. Regarding the pathological appearances found in the cortex, he said nerve-cell degeneration was as prominent as purely inflammatory changes, and operative measures could not influence it.

Dr George Wilson suggested that operation was likely to benefit cases in which there were localized motor symptoms.

Dr Clouston warmly congratulated the writers of the paper on the care and thoroughness with which they had done their work. It had given him great pleasure to hear Dr Macpherson's masterly pathological descriptions of general paralysis. He justified any kind of experimental treatment in this hitherto hopeless disease. He believed that our physiological and pathological knowledge had been increased by the facts brought out in the paper. As regards the question of pressure, he believed that there was no evidence that the intracranial pressure is increased in general paralysis in any stage, except, perhaps, after congestive attacks from vaso-motor paralysis and dilatation. He believed that pressure would tend to cause drowsiness, stupidity, and lethargy, rather than maniacal excitement. All the appearances could be explained on the theory of the fluid being compensatory. In regard to the question whether the disease was essentially an inflammation or a nervous degeneration, he was strongly of opinion that it was a degeneration accompanied by certain inflammatory or proliferation products the result of vaso-motor irritation and partial paralysis. The course of the disease is that of a typical nerve degeneration originating in the highest mental and motor centres, and passing downwards till it reaches the furthest ganglia and the peripheral nerves, involving the cord, retina, and sympathetic ganglia. The occasional high temperature, which is seldom an inflammatory temperature, is explained by the maniacal conditions and by the congestive attacks. The headaches are no doubt caused by meningeal congestion. And more than one-third of the cases

have no heightened temperature, or headaches, or maniacal excitement at all, but a simple progressive dissolution of mental and motor power. How can the universal vascular and lymphatic changes and degenerations all over the region of the two carotids, outside the cranium as well as inside, be explained on the intracranial inflammatory theory? The hæmatoma auris is most common in this disease, owing to such degeneration outside the skull, and pressure has nothing to do with that. The theory of vaso-motor changes explains all those phenomena. It is now well recognised that each cortical centre has its own vaso-motor and trophic centre contained within its own area, so that when a mental area or many such areas are affected, as in this disease, their vaso-motor centres are also affected. If the disease is inflammatory, or even essentially vascular or lymphatic in origin, how can the fact of uniform progression till death results in every case be explained? How is it that the ordinary fibrous and purulent products of inflammation never by any chance are seen in any one case? He had seen an ordinary meningitis superimposed on the general paralytic process, and the respective pathological changes of the two diseases were quite distinct. He did not agree with Lewis that vascular and lymphatic changes were seen before nerve-cell changes, and that they were found in a more advanced stage than the cell degenerations. Besides, he did not believe that by our present means of investigation we can as readily detect early nerve-cell changes as early vascular and lymphatic changes. Finally, the almost universal tissue and organ degenerations that are found in the end of the disease are explained more rationally on the theory of a nervous degeneration beginning in the highest nerve centres, and early involving most of the trophic centres. He regretted that the meeting had not this evening the benefit of the views of Dr Batty Tuke, who strongly upheld the intra-cranial pressure and inflammatory theories in this disease, on account of his being unavoidably absent in London on the service of the College of Physicians.

Dr Macpherson thanked the members who had criticised the paper for the kind manner in which they had referred to his own and Mr Wallace's work. He was particularly struck by the divergence of opinion expressed by the various speakers. This difference of opinion upon the pathology of general paralysis was sufficient evidence, if that were needed, to show how little was yet really known regarding the disease in question. A discussion like the present one was therefore useful in helping to clear the atmosphere of the many fanciful theories that still cling around our knowledge of the pathology of this fatal disease. Two main theories now hold the ground between them, namely, the degenerative and the inflammatory theories. The degenerative theory receives the weighty support of Dr Clouston. He must confess that if they had been fully convinced of the fact that general

paralysis was a true progressive nervous degeneration they would not have considered it advisable to undertake the operation they had just described. There were one or two points that seemed to them to tell strongly against the primary degenerative theory. They were—(1), the first appearance of the disease in the non-nervous tissues of the brain; and (2), the fact that remissions in the course of the disease occur in which all the symptoms, both mental and motor, may disappear. These remissions cannot be reconciled with the degenerative theory of the disease, and are most readily accounted for by the theory of the subsidence of the vascular inflammation. Both Dr Ireland and Dr Clouston spoke slightingly of pressure symptoms, and consequently of any operation for the relief of those symptoms. These gentlemen cannot, however, disregard the presence of fluid in abnormal quantities, which does not so much tend to cause coma as irritation, and the symptoms of early general paralysis are undoubtedly analogous to those produced experimentally by substances which irritate the cortex (*vide* Dr Dean's paper in the *Pathological Journal* for May 1892). Mr Wallace and he had pointed out, on the authority of Duret, the pia arachnoid sac is capable of containing very considerable quantities of fluid without causing coma. An objection had been brought forward against the operation by Dr George Robertson, who stated that the small quantity of fluid removed could have no influence upon the future course of the symptoms or the future accumulation of fluid. If the inflammatory theory of the disease is correct, then they could point to the exactly analogous operations for the relief of acute pleural effusions, and for the cure of glaucoma. He regretted the unsatisfactory nature of their results. He was not unhopeful that there might still be found some surgical means for the cure of general paralysis, and he wished Dr Batty Tuke every success in his decision to attempt the relief of that disease by the performance of laminectomy.

2. ON THE RESTORATION OF THE APPARENTLY DROWNED.

By William Alexander, M.D., Brigade Surgeon, A.M.D., Glencorse.

Death by drowning is the result of suffocation, either from the direct introduction of water into the air passages, or from closure of the glottis, or by a combination of both these conditions. To attempt resuscitation with any possibility of success, the tendency to closure of the glottis should be removed, thus giving an opportunity for any fluid to escape from the bronchial tubes, and afterwards for the admission of air to the lungs.

In a paper by Dr Howard, read in London Medical Society, October 22nd, 1888, it was demonstrated that the most effectual method of keeping the entrance to the larynx open during adminis-

tration of chloroform, etc., was by forcibly extending the neck backwards, which raised the valve. Shortly after this, when engaged in instructing a detachment of the Medical Staff Corps on the subject, it struck me forcibly that the simple procedure recommended by Dr Howard was equally applicable in cases of apparent death from drowning, and I became aware that the instructions laid down in certain official manuals were erroneous, and the illustrations on the subject misleading.

I have not access to the original descriptive teachings of Drs Sylvester and Marshall Hall on the subject, but in the manual before me there is room for improvement. The descriptions given in it are as follows:—

Dr Sylvester's Method.—"Place the patient on his back on a flat surface, inclined a little upwards from the feet. Raise and support the head and shoulders on a small firm cushion, or folded article of dress placed under the shoulder blades. Draw forward the tongue and keep it projected beyond the lips; an elastic band over the tongue and under the chin will answer the purpose; or a piece of string or tape may be tied round them, or by raising the lower jaw the teeth may be made to retain the tongue in that position." Then follows the extension movements. Any one who has tried to follow this description practically will be aware of the great difficulty, if not impossibility, of dealing with the tongue in such a manner, and the result of such procedure on the epiglottis is at least problematical, and many years ago Sir Joseph Lister showed that this method of raising the epiglottis was inefficient.

Dr Marshall Hall's Method is more scientific, and is thus described:—"Place the patient on the ground face downwards, and one of the arms under the forehead, in which position all fluids will more readily escape from the mouth, and the tongue itself will fall forward, leaving the entrance to the windpipe free," etc. It appears to me very doubtful if this position will forcibly counteract the spasmodic closure of the glottis, but with the body placed on an inclined plane, feet raised and the head more forcibly extended backwards, there would be a better chance for establishing artificial respiration.

In my lectures of instruction on this subject I have been in the habit of teaching a combination of the two plans, Sylvester's and Marshall Hall's. Having first placed the patient on his back on an inclined plane with *the feet raised*, and the head thrown forcibly backwards, which insures the raising of the epiglottis, thus affording a means of exit to any fluid in the air tubes, and likewise a means of entrance for air when artificial respiration commences; with an assistant to steady the feet, the forearms are seized and forcibly extended above the head, then brought down suddenly to side, a hand of each operator at the same time pressing downwards the ribs in front; the patient is then rolled over, taking

care that the face is not injured, then brought back quickly to original position. This is to be repeated 18 times to the minute, and always to the same side, till natural respiration is established, as it seems in accordance with reason that if water has invaded the lungs, it would give the person a better chance of resuscitation to thus clear one lung by gravitation and expulsion, and when life was restored then to vary the position. Warmth should, of course, be promoted by friction and hot flannels if available, care being taken not to give the *coup de grace* by pouring brandy or other alcoholic drink into the mouth before the power of swallowing is re-established.

There is nothing new in the method I propose except in the application of previous methods. The subject was fully explained, so far back as November 1863, by Dr Robert L. Bowles, yet practices which were then and subsequently demonstrated as impossibilities have been allowed to reappear in our text-books all these years. It is in the hope of improving the teaching of this subject that I venture to bring it forward again in 1892.

Dr James Ritchie said that doubtless the Howard method of extending the head upon the neck was not only the best, but the only way of insuring a free opening for the passage of air during artificial respiration. He asked Brigade-Surgeon Alexander whether he had found it possible for a single individual to carry out the modification of the Hall and Sylvester methods which he recommended, at least for a length of time. By the Howard method it was quite possible for a single operator to continue the resuscitating process for a long period without fatigue.

Brigade-Surgeon Alexander, in reply, said that the main object he had in view was to call attention to the obviously erroneous teaching on the subject in our various text-books, manuals, and the instructions issued by the Royal Humane Society, and taught by the Ambulance Societies all over the country. The modification he proposed could not be carried out for any length of time by a single individual, but assistance was generally at hand in these cases.

Meeting X.—June 1, 1892.

Mr Joseph Bell, *President, in the Chair.*

I. Exhibition of Patients.

1. *Dr John Thomson* showed—(*a.*) One of the cases of congenital laryngeal stridor about which his paper was written; unfortunately it was the least typical of his cases. The infant, a girl,

aged eleven weeks, had been noticed to breathe in a noisy way since a week after birth. The "stridor" was less loud than in the other cases seen, and the intermissions were longer and more frequent. The noise was, however, fairly typical when it was present, and it varied under different conditions just as in more marked cases.

(*b.*) A little girl, aged a year and eight months, with UNEQUAL DEVELOPMENT OF THE LOWER EXTREMITIES, the right lower limb being a third of an inch longer than the left; the right thigh one inch, and the right calf one quarter inch larger in circumference than the same parts on the other side. The child was otherwise remarkably well developed, and the rest of the body quite symmetrical. The difference was first noticed about a year ago. The temperature, consistence, strength, and reflexes of the two lower limbs seemed the same. The case was regarded as probably one of simple hypertrophy of the right limb.

2. *Dr Shaw M'Laren* showed a TUMOUR of the SCAPULA, and the patient from whom it had been removed.

II. Exhibition of Specimen.

Dr P. M'Bride showed a TUMOUR removed from the anterior extremity of the right vocal cord.

III. Exhibition of Preparation.

Dr W. Allan Jamieson showed specimens of various soaps, and remarked, that in an age when the word "Soap" meets the eye at every turn, and when a well-known firm of soap-makers could announce that they had spent in one year more than £120,000 in advertising, it was especially needful that the kind employed should be that which is suited to the skin. It has been shown that many of the soaps in common use are alkaline ones, and as such are injurious. The superfatted soap introduced by Unna is excellent, yet there is no good reason why we should have to go to Germany to get our soap. At my suggestion Messrs Duncan & Flockhart have prepared, under the able direction of Mr D. B. Dott, a soap which is certainly superior to any superfatted soap which I have tried. This, registered as "*Baumol superfatted skin soap*," is made of the purest fat, contains no cocoa-nut oil, nor any objectionable ingredient, and is not only perfectly neutral, but is superfatted to the extent of three per cent. It will be found to keep well, to be most pleasant in use, reasonable in price, and for all toilet purposes perfect, but "it won't wash clothes." As shaving has again by revolution of time come into fashion, Messrs Duncan & Flockhart have prepared a suitable stick. But a soda soap like this does not cleanse the skin as one with a potash basis does. I have here a superfatted potash soap, which in diseased conditions demanding the employment of a soap to remove adventitious products leaves nothing to be desired. It cleanses the skin, yet after its use the

surface remains soft, smooth, and polished. It is a semi-solid soap, but Buzzi has lately drawn attention to fluid soaps, and here are two fluid soaps prepared by the same firm—one neutral, one superfatted with lanoline. The neutral is clear and limpid, the superfatted a trifle cloudy. Should an alkaline soap be desiderated, then one can with ease be prepared by adding four per cent. of pure carbonate of potash, and thus a soap of definite and mild alkalinity is obtained. Such an one resembles Hebra's alkaline soap spirit, but possesses many advantages over that rather crude compound. These soaps have the superiority over solid ones that an exact quantity can be used. They are serviceable in carrying out the new soap treatment as contrasted with the old. The old consisted in removing by a strong alkaline soap all the crusts, scales, or debris, but along with this the lubricating fat of the skin, then replacing the latter by an astringent and oleaginous salve. The modern consists in incorporating with a neutral or superfatted soap—hard, soft, or fluid—the desired ingredient, and applying this, plain or diluted, to the skin. As an example I have here a fluid tar soap, which promises to be a valuable adjunct to our armamentarium in the treatment of psoriasis. For an infinity of other kinds I must refer you to the writings of Unna, Eichhoff, and Buzzi.

IV. Original Communications.

1. SOME QUESTIONS WITH REGARD TO TUBERCULOSIS OF THE UPPER AIR PASSAGES.

By P. M'Bride, M.D., F.R.C.P. Ed., F.R.S.E., Aural Surgeon and Laryngologist to the Edinburgh Royal Infirmary ; and Lecturer on Diseases of the Ear and Throat, Edinburgh School of Medicine.

The pathological identity of lupus and tuberculosis can, I think, be no longer disputed. To anyone who even cursorily investigates modern literature on the tubercle bacillus, it must be evident that this organism is responsible for both diseases, and we must therefore admit that lupus and tuberculosis are but manifestations of the same process. Thus the microscopic structure is almost, if not quite, identical; diffuse tuberculosis has been produced from the inoculation of lupus tissue; a skin disease having all the characteristics of lupus has been observed to occur in the neighbourhood of cutaneous orifices leading from deep tubercular foci; lupus reacts to Koch's tuberculin; and, finally, many patients suffering from the cutaneous malady also present other evidences of tuberculosis. While, therefore, now quite convinced that lupus is to all intents and purposes tuberculosis, I shall still, in the course of these remarks, retain the term with the view of making clear the points which I desire to bring out. Indeed, the clinical distinctions between what has always been, and is still, described as lupus of mucous membranes and what is termed tuberculosis

are often so marked that it would not be wise to abandon the designation.

It is somewhat interesting to compare in a general way the manifestations produced by the tubercle bacillus in the skin and in the mucous membrane of the upper respiratory tract respectively. When the organism attacks a dermoid surface it commonly produces the condition known as lupus, but in very exceptional instances true tubercular ulcers are formed, more particularly in parts where skin is approaching mucous membrane, but sometimes in other areas, such as the arms and legs. When the anterior nares are attacked true lupus is probably more common than typical tuberculosis. The former is characterised by the presence of a nodular infiltration resembling in some respects granulation tissue, while the latter manifests itself either as a tubercular tumour or an indolent ulcer. Both forms commonly attack the septum, but in exceptional cases the inferior turbinated body, and probably other parts also may be first affected. It has been my fortune to see a considerable number of cases of lupus of the nasal mucosa, some primary, but most of them associated with the presence of the eruption in other parts. It has appeared to me that the disease is often considerably modified by situation. Thus near the nasal orifice one sees a nodular infiltration, presenting here and there a yellow transparent colour, the corresponding portions being at the same time very soft when felt with a probe, which can easily be made to penetrate them. These parts appear to have a tendency to become crusted over, but I have not usually been able to associate this scabbing process with any definite macroscopic ulcerative breaking down. As the disease is traced inwards it is found that the nodules are more distinctly red and have a much firmer consistence. I cannot recall any very definite ulceration here either. Sometimes the nodular surface is tinged by a pus-like secretion which indicates that ulceration is in progress, but the process is of infinite slowness, and on cleansing the surface its source is not generally apparent. Such, then, has been my personal experience of lupus involving the nasal mucosa, and I shall venture to bring before you a case of some interest which, excepting for the location of the disease, may be taken as fairly typical.

A young lady was brought to me by Dr Burn-Murdoch on account of nasal obstruction, specially marked on the left side, which had lasted for some years, but had become worse during the last six weeks before her visit, early in the spring of this year. On examining the anterior nares the left inferior turbinated body had an eaten-out appearance, beginning near its anterior extremity. The surface of the affected part was distinctly nodular, there was no trace of purulent secretion, but it bled rather readily when touched with a probe. In the opposite nostril there was a nodule covered by a scab in front of the inferior turbinated body.

It is not my purpose in this paper to refer to treatment, but I must mention that the diseased part was scraped out and microscopically examined. It consisted of granulation tissue containing in its substance many epithelial cells, but no true giant cells or bacilli were detected. A small portion of the tissue was, however, handed over to Mr Alexis Thomson, and he succeeded in producing tuberculosis in a guinea pig by inoculation with the lupoid tissue. I may perhaps also add that to all appearance the young lady was typically healthy, there being no evidence of pulmonary or cutaneous disease. The diagnosis in this case was arrived at by a consideration of the similarity in appearance between the affected mucous membrane and the condition found in undoubted cases of lupus where the cutaneous affection was also developed in its typical form. The eaten out appearance presented by the left turbinated body, it may be said, gave definite proof of the occurrence of ulceration. Here, however, I am rather inclined to join issue, for it seemed to me rather to indicate submucous absorption permitting falling in of the lupoid surface. Indeed, from my clinical observations on lupus of mucous membranes, I am rather inclined to think that such limited destruction of parts as occurs is often due to a caving in of the nodular surface rather than to external breaking down.

Let us now glance at the lesions liable to be produced in the pharynx by the tubercle bacillus. By the term pharynx I mean the throat as examined by the tongue depressor, and do not include the naso-pharynx, where too few tubercular lesions have been observed to admit of any generalisations. Moreover, I have intended this paper to be rather a brief account of individual experience than a digest of the observations of others, and I have with one doubtful exception never seen tuberculosis in this region.

In the pharynx proper I have met with at least two distinct varieties of tuberculosis, always associated with pulmonary disease, and characterised by marked pain.

In one form the palate and, indeed, the whole pharynx are extremely pale, here and there ulcers are seen of varying size; these may be situated on the tonsils, the posterior pharyngeal wall, and the soft palate. These ulcers are all shallow, covered with muco-purulent secretion, and their floors have a tendency to produce pale pink granulations. In advanced cases the whole posterior wall of the pharynx may be converted into one large ulcerating surface, the yellow floor of which is relieved by protruding granulations. The second form of pharyngeal tuberculosis I have met with only once in a lady who, while showing little evidence of pulmonary disease, suffered from loss of voice. Examination of the larynx demonstrated marked œdema of the epiglottis and the characteristic pyriform swelling of the right ary-epiglottic fold. At the first visit the uvula and right lateral fold of the pharynx were œdematous, of a grayish colour, like that

seen in tubercular infiltrations of the larynx, and studded with small yellow nodules, which I assumed to be miliary tubercles. Some six months later, after a residence at Bournemouth, the pharynx was rather better, but the larynx remained as before.[1]

Of lupus of the pharynx, either primary or associated with a similar condition of the larynx, I have seen several cases. In these the area chiefly or entirely attacked has commonly been the soft palate. This part is studded over uniformly with small red nodules, but I have never seen ulceration to any marked extent, although I have observed it where the hard palate is involved. The lupus nodules in this situation seem to me to be of much firmer consistence than are those found about the anterior nares. From the description I have attempted to give it will be evident that lupus and tuberculosis can be clinically distinguished from each other in this region. As to the frequency of tuberculosis and lupus of the pharynx, both are extremely uncommon, and I am not aware that any statistics exist as to the relative numbers of observed cases of each disease. Judging from my own experience, I should say that one is met with as often as the other.

If we now pass to the larynx we find a very different state of matters. Here, as everyone knows, tuberculosis is quite common, while lupus is relatively rare. It is true that observers who have not confined themselves to laryngeal clinics have found the larynx affected in from 5 to 9 per cent. of lupus patients where these were systematically examined; notwithstanding these results it must still be admitted that laryngeal lupus is far from common.

In the cases I have observed the epiglottis, the false cords, and the ary-epiglottic folds have been attacked. These parts showed a nodular infiltration of red colour, and there seemed again to be the same absence of superficial ulceration. The disease differs from phthisis laryngea in almost every detail. The colour of the thickened parts is red in lupus, it is usually pale in phthisis; the surface is commonly nodular in one (lupus), it is smooth in the other. The course of typical tuberculosis is rapid, *i.e.*, the patient generally dies within two or three years. It is slow in lupus; thus I heard recently that a patient whose larynx presented marked lupoid changes in 1883 is still in good health, and that her throat is better than it was. Lupus has often little or no tendency to superficial ulceration, while in tuberculosis this tendency usually soon shows itself. I have, I think, said enough to prove that it is not only perfectly fair but necessary to make a clinical distinction between lupus and tuberculosis of the larynx.

[1] This patient was again seen since this paper was read. The pharynx has lost all traces of disease; the epiglottis, too, has almost returned to its normal dimensions. There still remains, however, an infiltration of the right ary-epiglottic fold. The treatment was general and local (menthol and lactic acid applied by Dr Thomson of Bournemouth and myself).

Let us now consider how far we are entitled to draw any general deduction from the facts before us. We have seen that the skin is but rarely attacked by true tuberculosis, but frequently by lupus; we have further seen that in the larynx tuberculosis in its typical form has an immense numerical preponderance over lupus. Further, it is probable that lupus is distinctly more common in the mucous membrane at the nasal orifices than tuberculosis, and that in the pharynx the two conditions are about evenly balanced. I doubt, however, whether without exact statistics it would be right to attach too much importance to the two last-named points. Without them, however, merely considering the skin on the one hand and the larynx on the other, we have enough evidence to suggest that the tendency of the same organism to produce a lupus manifestation in one situation and a tuberculosis in the other is probably due to anatomical differences in the parts affected. The nature of these differences is, however, anything but clear. Other factors besides histological distinctions probably play a part, for the mucous membranes of the upper air passages are under physical conditions which are by no means similar to those of the skin. Although the presence of lupus and tuberculosis in mucous and dermoid surfaces respectively are, from a consideration of the facts adduced, in all probability to be accounted for on anatomical grounds, yet there are difficulties in accepting this view as even a sound working hypothesis. Individual predisposition must also be admitted to play some part, for on what other supposition can we account for the comparatively common occurrence of laryngeal lupus in those who are the victims of the cutaneous malady, while primary laryngeal lupus or lupus secondary to pulmonary disease is admittedly of extreme rarity? With regard to the question of position as influencing the occurrence of lupus or tuberculosis in its more typical form, it is of interest to note that lupus has rarely, if ever, been known to extend into the trachea. I am not aware whether any lesion corresponding in its macroscopic characters to lupus has ever been described by pathologists as occurring in the respiratory tract below the upper portions of the windpipe. In the digestive and deeper genital mucous membranes I do not know that such a disease has been found; it has once been recorded as observed in the middle ear, while it has not been met with in internal organs. Of course, in considering this aspect of the question it must be remembered that the difference between lupus and tuberculosis is chiefly macroscopic and clinical, and that therefore the differentiation could not be carried out in parts not accessible during life. I need hardly say that I have consulted older works written at a period when the two conditions were considered as distinct, with a view to the elucidation of the question just raised; but even in these (edition of Klebs' *Handbuch der Patholog. Anatom.*, 1873) I find no reference to lupus of the parts referred to, *i.e.*, internal

viscera. As a rule, the macroscopic difference between lupus and tuberculosis of the mucous membranes of the upper air passages is so marked, that looking at the question from a purely clinical point of view, it is extremely difficult to believe in the identity of the two diseases. I am, of course, aware that ulceration has been described by many authorities (*e.g.*, Neumann, *Atlas der Hautkrankheiten*) as a common feature of lupus of mucous membranes. That it may be so I do not deny, but in that case my own experience has been of an exceptional nature, and, as before mentioned, it is my desire to introduce into this discussion rather what I have observed than to record the observations of others. The position I should be inclined to take up with regard to the ulceration of lupus in mucous membranes is as follows:—The parts affected by the infiltration are always irregular and thickened, therefore they are exposed to injury from contact with adjacent parts or extraneous substances; as a result of such injury ulceration may take place, but breach of surface is not a direct or early result of the diseased process as in tuberculosis. I have no desire to insist upon the accuracy of this view, but only to state that a comparatively limited series of clinical observations has inclined me to adopt it.

We have seen, then, that there is a marked clinical distinction between the two forms of disease as produced by the action of the tubercle bacillus. We have also seen that all recent observations have tended to show that this organism is responsible for both varieties. It remains to consider whether transition forms are met with. Again, relying merely upon my experience, it appears to me that such forms occur, but that they are of extreme rarity. So long as I could not either adduce or find described such transitional stages, it seemed to me that a very serious link was wanting in the chain of argument which seemed to prove the identity of lupus and tuberculosis, although I am not prepared to say that these arguments would be invalidated even by the absence of intermediate conditions. I can, at present, only recall three instances in my own practice. Of one I can only write from memory, as owing to the circumstances of the case no notes were kept; of another I have a fairly complete record; while the diagnosis in the third instance admits of some possible doubt.

The first case was that of a young gentleman of distinctly strumous habit, showing glandular enlargement about the neck, and, if I remember right, cicatrices in the same situation. He had aphonia some five years or thereabout before his death, and when I first saw him the larynx was in a state of advanced disease—infiltration of the epiglottis, ary-epiglottic folds, and ventricular bands. There was distinct stenosis due to this cause, while although there was evidence of pulmonary disease, this was not found to be either very active or very advanced. To show the slow nature of the malady, it may be mentioned that long after

his visit to me the patient was able to ascend on foot one of the highest mountains in Scotland. The laryngeal infiltration differed from that usually seen in being of a red colour, in having a more uneven outline, and by evincing but slight tendency to ulcerate. Unfortunately, owing to the absence of notes I cannot lay much stress upon this case. In the case of the other patient, however, I have fairly full notes, especially with regard to the point at issue.

A. B., æt. 17, consulted me at the Infirmary in 1888. On examining the larynx the epiglottis was seen to be much thickened, of red colour, and nodular. Both ary-epiglottic folds were much infiltrated, especially the right. The posterior wall of the pharynx was marked by distinct ridges, the uvula was somewhat thickened, with yellow nodules embedded in its substance. The submaxillary and cervical glands were swollen, and, according to the statements of the patient and his father, had always been so. There was a small patch of skin eruption on the left side of the chin, but to the best of my recollection—here I write from memory—this was not lupus. On examining the lungs prolonged expiration was found at both apices, and some impairment of percussion at the right base. There was at this time no cough, and "no hæmoptysis" is recorded in my notes. The throat only got bad two months before my examination; the symptoms complained of were hoarseness and pain on swallowing. There was no dyspnœa of any consequence. I lost sight of the patient for some time on account, I think, of my questions with regard to the possibility of specific disease. I, however, saw him somewhere about a year afterwards, when the larynx was, so far as I remember, in much the same condition as before. The appearance of the palate had, however, changed. Whereas at his first visit there were small yellow points quite like those already described in a case of undoubted pharyngeal tuberculosis, there was then a very distinct uniformly red nodular infiltration extending over the soft palate.

I after this lost sight of the patient for a time, but about the period when tuberculin was being so largely employed he came back to me. There was then very marked laryngeal stenosis,—indeed, his case is the original of the drawing of laryngeal lupus in my recently published work. The epiglottis and false cords were so infiltrated that the true cords were invisible and the respiratory lumen much narrowed. Realizing that active treatment was necessary, and considering that the risks of tuberculin would be less than the dangers incurred by either endo-laryngeal scraping or the same process after thyrotomy, I suggested the employment of Koch's method. Prof. Chiene very kindly took the patient into his ward—as I had then no beds—and carried out the injections. He thought it desirable to perform a preliminary tracheotomy, and I feel sure that this was a wise step, in so far that it prevented aspiration of diseased tissue and removed all danger from dyspnœa. The patient only reacted with difficulty—after several injections—

and after a time these were given up, because it was thought they were doing no good. Only some weeks later did a marked improvement occur, and on laryngoscopic examination, I was, to my surprise, able to see both vocal cords, which showed as white bands—these having evidently escaped lupoid infiltration. I do not know the future history of this patient, but no doubt marked temporary benefit followed the employment of tuberculin. This was the only case in which I ever felt justified in distinctly advising its employment, for the only alternative was a surgical operation of such a character that it would have directly endangered the life of the patient.

The third case was that of a boy of fifteen, whom I first saw in December last, and full details of which will, I hope, be published at some future time by Dr Gemmell of Airdrie, whose patient he is. The history pointed to a throat affection having lasted about four years; this went on, in spite of treatment, until his visit, when he complained of difficulty in swallowing and a pain in the roof of his mouth. On examining the throat the following points were noticed. The hard palate presented marked nodular infiltration, which was and is still extending over the gums. The pharynx presented no signs of active disease, but had all the appearances of healed ulceration, so commonly met with in specific cases: there was marked stenosis, the uvula had disappeared, and a thick mass corresponding in position to the left lateral fold partly occluded the passage. With the laryngoscope the epiglottis was seen to be thickened on the left side. This thickening has now assumed a nodular character, while the corresponding ary-epiglottic fold presents a pyriform swelling more like typical tuberculosis. There was thickening of both alæ nasi at the patient's first visit, and externally a nodule not unlike lupus was noticed. Both nostrils have since become occluded by masses of thickened mucosa, which according to my notes have no definite nodulation. The patient, notwithstanding his ailments, had a remarkably fine physique, the teeth were not notched, there were some enlarged glands at the side of the neck, and a patch of doubtful dulness over the right apex. It need hardly be said that iodide of potassium was given, and at one time mercurial inunctions were advised. The latter seemed, however, to have a distinctly harmful influence, for just afterwards the nodules in the palate began to ulcerate. On ceasing the use of the mercury these ulcerations rapidly healed, and the nodular infiltration only was apparent. If, as I am inclined to think (and I believe Dr Gemmell shares this view), this is purely a case of tuberculosis, the process must have entirely healed in the pharynx and broken out or persisted at three distinct points—to wit, the anterior nares, the hard palate, and the larynx, where with the exception of arytenoid region it had the clinical characters of lupus. During the temporary ulceration

which was apparently caused by the mercury, some of the secretion of the mouth was examined for bacilli with a negative result, so that absolute proof of the tubercular character of the disease is wanting, but the negative effect of potassium iodide and the clinical features make it almost certain that the throat affection is partly, if not wholly tubercular.

In concluding this paper I must apologise for having appeared to ignore literary references. I have avoided them, except very incidentally, because it has been my desire to bring forward merely my own experience, which as to certain points, *e.g.*, the frequent absence of any marked tendency to ulceration in lupus of mucous membranes, seems to have been somewhat out of accord with the descriptions of most writers, and to obtain the verdict of members of this Society as to which view tallies with general experience.

Dr W. Allan Jamieson congratulated Dr M'Bride on his remarkably able and lucid paper. Personally he had comparatively little experience of tubercular affections of mucous surfaces. He had seen the hard palate sometimes, the gums, especially of the upper jaw, frequently attacked by lupus. In the latter region there were apt to be exuberant granulations and superficial ulcers. The condition was an obstinate one, the unhealthy secretions of the mouth combined with the disturbance caused by mastication baffled efforts at cure. Tuberculosis of the skin, however, offered many analogies to that of the mucous membranes. Miliary tuberculosis of the skin was very rare in his experience. It tended to ulcerate rapidly; this he thought might be due to the fact that the bacilli were abundant as compared with lupus. Their quantity rather than their quality seemed the chief cause of the differences which undoubtedly existed between miliary tuberculosis and lupus of the skin. Lupus vulgaris, though met with elsewhere, was found chiefly primarily on exposed parts, and on regions which were the most vascular,—the face, hands, and feet. There the bacillus had a more congenial soil than in situations where the skin was normally more anæmic. Ulceration did not occur unless there was solution of continuity on the surface, and then was due to the access of pyogenic organisms. Lupus verrucosus was the most superficial of the forms constituting the lupus family, then came lupus vulgaris, and, lastly, the tuberculous gumma commencing deep in the substance of the skin. Of all varieties the warty was the one most plainly due to local infection. It was seen in washerwomen, in those coming in contact with tuberculous sputa from phthisical patients, etc., starting in such often on the knuckles, and so apt to fissure.

Dr M'Kenzie Johnston said that he thought Dr M'Bride's paper one of very great interest, and one which supplied much food for thought. He entirely agreed with all that was said in it as to the

identity of lupus and tuberculosis, and also as to the well-marked clinical differences exhibited by them, which, doubtless, were sufficient reason for maintaining the nomenclature as at present. He had been much interested in Dr M'Bride's remarks on the frequent absence of essential ulceration in lupus. This was not quite in accord with the views of other writers, and he should like to reserve his judgment for the present on this point. In future he hoped to examine his cases while keeping this suggestion in mind. With reference to the greater frequency of lupus in the nose and of tuberculosis in the larynx, Dr M'Bride had suggested that this might be due to anatomical differences in the regions under consideration. He would like to offer as a suggestion, that it might be due to physical conditions affecting the multiplication of the bacilli rather than to anatomical conditions. In the larynx, the moisture and warmth, and the difficulty of getting rid of the secretions, were just those conditions which facilitated the growth and multiplication of the organism, and it was in this region that lupus was most rare and tuberculosis relatively most frequent. In the nose, on the other hand, the free play of the atmosphere had the effect of keeping the part dry and cooler, and the greater ease with which the secretions are dried up or removed in this situation is unfavourable to the growth of the bacilli, and in this region lupus is relatively most common. He therefore suggested that the nearer the surface and the more exposed the part is to the atmosphere, the greater would be the chance of the appearance of lupus rather than of tuberculosis. As mentioned by Dr Jamieson, in lupus the bacilli are numerically fewer than in tuberculosis, while in other respects similar, and this would seem to lend some support to his theory.

Dr M'Bride briefly replied, and thanked the members for the kind way in which his paper was received.

2. ON INFANTILE RESPIRATORY SPASM (CONGENITAL LARYNGEAL STRIDOR).

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Infantile respiratory spasm, or congenital laryngeal stridor as it is sometimes called, is a peculiar form of obstruction of the respiration occurring in very young infants. It is not often met with in its most marked forms; it is not of serious import for the child's health; and it is not amenable to any special treatment. These considerations may perhaps account for the somewhat surprising fact that the condition is not even mentioned in most of the text books on diseases of children or laryngology, and is nowhere fully discussed. It is, nevertheless, well worthy of

attention from a clinical point of view, because it is apt to cause anxiety owing to its being mistaken for some more serious disease; and also from the point of view of pathology, because its causation is not as yet thoroughly understood.

In the few references to the condition in medical literature which I have been able to find, a different name has been used in describing it almost every time. Thus, Dr D. B. Lees,[1] describing the post-mortem appearances of a case, speaks of it as a "Peculiar Form of Obstructed Respiration;" Dr Gee[2] calls some cases which seem to be of the same nature (although in some respects different) "Respiratory Croaking;" and Dr Goodhart[3] refers to the ailment as "Infantile Laryngeal Spasm." Again, Dr Suckling,[4] on showing a case to the Midland Medical Society two years ago, called it "Congenital Laryngeal Stridor;" Dr Löri of Pesth[5] uses the term "Clonic Spasm of the Glottis;" and Dr Robertson[6] has described one case apparently of this nature, along with others, under the designation "Posticus Paralysis."

Probably the best of the names is that used by Dr Goodhart, but it is perhaps preferable to call the affection "Respiratory" rather than "Laryngeal" spasm, as it seems doubtful whether there is any more spasm of the laryngeal muscles than there is of the other muscles involved in the respiratory act. "Congenital Laryngeal Stridor" is also a good name, and has the advantage of marking more distinctly than the other the clinical difference between this ailment and ordinary laryngismus.

During the last two or three years, I have had the opportunity of studying five well-marked examples of this affection, as well as a few other milder cases apparently of the same nature. In the present paper I propose to draw attention to the clinical histories and symptoms of these; to the data which they afford for determining the pathology of the condition; and to the conclusions that may reasonably be drawn from them.

The following are notes of the five most characteristic cases:—

Case I.

John K., aged 4 months, brought to the Sick Children's Hospital on 7th April 1890, because of noisy breathing. Both parents are strong and healthy; their only other child, a boy, is quite normal and healthy. The mother was quite well during her pregnancy, and the labour was easy and natural. The child has been on the breast and has thriven well.

When about a fortnight old he took a "cold," and his mother thinks

[1] *Trans. Path. Soc. Lond.*, xxxiv. p. 19.
[2] *St Bart. Hosp. Rep.*, xx. p. 15.
[3] Goodhart, *Diseases of Children*, 1885, p. 251.
[4] *British Med. Journ.*, 1890, i. p. 607.
[5] Löri, *Allg. Wiener med. Zeitung*, 1890, No. 49.
[6] Robertson, *Journal of Laryngology*, Oct. 1891.

that it was at that time that the noisy breathing began; at least she does not remember it before. The noise is rather less loud now, on the whole, than it used to be, but it varies a good deal from time to time. It is said to be worse if he is excited, and also if he has any cold. He is thriving very well in other respects, and has nothing else the matter with him except a scurfy condition of the scalp and occasional dyspepsia with green slimy motions.

The patient is a big, but rather pale and flabby child. There is no evidence of rickets and no malformation of any kind, and the heart and abdominal organs are normal. There is a strip of impaired percussion at the base of the right lung, otherwise the chest is normal. The respirations are 40 per minute (pulse 140), and they are accompanied by a loud noise which varies in intensity from time to time. The inspiration begins with a hoarse sound, and when the noise is at its loudest, ends in a sort of squeak or crow, which the mother well describes as "just like a hen." Expiration is accompanied by a shorter grunting sound. The cry is quite clear and natural. There is considerable indrawing of the lower antero-lateral aspects of the thorax at each breath, and the chest seems permanently narrowed in that region. On auscultation, the stridor is heard very loudly all over the chest. The fauces and tonsils are red and swollen from chronic catarrh. Dr R. W. Philip kindly examined the larynx along with me; only a partial view of it could be obtained, but so far as it was seen it appeared normal. Advice was given about diet, and a tonic prescribed.

21*st May* 1890.—The stridor is a good deal less loud and the periods of intermission much longer. The child is bigger and stronger; he is perfectly intelligent, although rather phlegmatic.

Dec. 1892.—Since the last note the child has been living in the country. The stridor is said by his parents to have disappeared about six months ago. Latterly it was only heard when he was annoyed or excited. From his mother's account, he is evidently very rickety.

Case II.

Harry W., aged 6 weeks, sent to the Sick Children's Hospital on 7th March 1891 by Dr Macdonald Robertson, on account of his peculiar breathing and for wasting. His parents, who are German Jews, are healthy, and he is their first child. The mother was well during her pregnancy, and her labour was easy and very short.

At birth, the child breathed in the same way as he does now, but the noise was not so loud; it has gradually got worse. It is not always equally loud, and sometimes ceases altogether when the child is awake. When he is asleep it is sometimes present, sometimes not.

The infant is very small and emaciated. He is on the breast,

and has some dyspeptic symptoms due to injudicious feeding. There is no congenital abnormality and no sign of rickets. The heart and the abdominal organs are normal. The percussion note over both bases is slightly impaired, but otherwise the lungs seem normal. The fauces are normal. Each inspiration is accompanied by a loud crowing sound, and there is also a slight noise with expiration. With each inspiration there is an exceedingly marked indrawing of the lower antero-lateral regions of the chest-wall and epigastrium, and also of the episternal notch. The child, however, does not seem at all distressed by dyspnœa, and there is no cyanosis. The cry is clear and strong.

2*nd March* 1892.—The child has steadily improved during the last year. He seems well-grown, well-nourished, and very intelligent. The noise with the respiration is much less loud, and the free intervals are much longer and oftener. When the stridor is present, the inspiratory sound is a sort of grunt or croak, and the expiration is accompanied by a blowing noise which is scarcely audible at a little distance. There is now only very slight inspiratory indrawing of the lower parts of the chest. There is some beading of the ribs and a certain amount of pigeon-breast. Dr M^{c}Bride kindly examined the throat. He found the fauces and upper part of the larynx absolutely normal, but was unable to get a good view of the vocal cords.

Case III.

Jeannie C., aged 11 weeks, brought to the Sick Children's Hospital, from the country on 22nd Oct. 1891, on account of her peculiar breathing and because she was not thriving. Both parents are said to be healthy, but the mother is "not strong" and looks anæmic. The patient is their seventh child. The fourth and fifth children died "from weakness" within a few days of birth; none of the family have suffered from any peculiarity of breathing. Labour was short, easy, and natural. The child has been given nothing but breast-milk; she frequently vomits, bringing up phlegm along with the milk.

The peculiarity of the breathing was noticed immediately after birth, and has remained about the same ever since. The noise disappears usually during sleep.

The infant is small and emaciated, but beyond the abnormal respiration shows no sign of disease or deformity. The cranium, heart, lungs, and abdominal organs are normal. The sternum is bulged forwards owing to the prolonged obstructed breathing. The respirations are usually about 40 per minute (pulse 140). The fauces, pharynx, and epiglottis seem quite normal. The vocal cords cannot be seen.

The inspiration is very noisy, beginning with a low pitch, but ending in a loud crowing sound like that of croup. The expiration is sometimes accompanied by a short grunting noise, but is usually

inaudible. The loudness of the sound varies from time to time. When it is at its loudest, the child's attitude is somewhat peculiar. She lies with her elbows out from her sides, her arms, wrists, and fingers being all much flexed and rigid. Her parents have noticed this attitude. The sterno-mastoids are seen to work strongly with each breath, and there is *very great* retraction of the episternal notch, so that it is alternately a slightly bulging prominence and a hollow fully half an inch in depth. The lower antero-lateral regions of the chest and the epigastrium are also retracted to an extreme degree with each inspiration. On looking at the violent movements of the muscles of respiration, and listening to the loud stridor, it is difficult to resist the idea that the infant must be in great distress; but on looking at her face, one notices that there is no cyanosis, that the alæ nasi are scarcely moving at all, and that the infant seems contented and happy, and is interested in what is going on around her. When she is watched for a little, it is noticed that she occasionally takes a full breath without any obstruction. The cry is very slightly hoarse.

27*th Nov.*—The child looks a little stronger; the breathing is very much as before; the cry is quite clear. Dr M^{c}Bride kindly examined her, and reports that the pharynx and the epiglottis and the rest of the larynx, so far as can be seen, appear normal, but that the larynx cannot be properly examined owing to the tendency to vomiting—milk from the stomach flooding the larynx whenever an attempt at examination is made.

24*th Dec.*—The general nutrition of the child is much better, but she has now a distinct rickety rosary. The breathing has much improved; the indrawing of the episternal notch and of the lower parts of the thorax is much less. The stridor is the same as before in character, but is not nearly so loud; the intermissions are longer and more frequent. Even when the breathing is noiseless, there is slight inspiratory retraction of the chest wall. The child often starts during sleep, and when she does so, she puts her hand up to her mouth.

April 1892.—The patient is much stronger, the stridor is less loud, and the free intervals longer. The retraction of the chest wall is also less.

Case IV.

James T., aged 11 weeks, sent to the Sick Children's Hospital on 5th March 1892 by Dr David Davidson on account of obstructed breathing. Both parents are healthy, and the patient is their first child. The mother was well during her pregnancy, but her labour was difficult, and chloroform and instruments had to be employed. The child is on the breast and has nothing wrong with him except the stridor, and he has always thriven very well.

The noisy breathing was noticed immediately after birth, but at that time it was not so loud as it is now. When the child is sound

asleep the noise ceases, but when he is not "very sound" it goes on continuously as during waking, with occasional short pauses and marked variations in intensity.

The patient is a very well developed healthy baby. There is no sign of rickets or of disease or malformation of any organ; and even when the noise is loudest there is no cyanosis. Nor is there any visible movement of the alæ nasi. There is no "facial irritability." The palate is highly arched. The fauces and epiglottis are normal.

The stridor is almost constantly present, but occasionally it ceases entirely for one or two minutes. It varies markedly in intensity from time to time. The more excited the child is, the louder is the noise; but, if he cries outright, the inspiration becomes almost noiseless. The cry is clear, loud, and long. The inspiration begins with a croak and ends with a high-pitched crow ("like a hen"). It is usually interrupted (being composed of two parts with a sort of jerk between). Expiration is accompanied by a short croak when the noise is loud; at other times it is almost, or entirely inaudible. The child seems quite happy and at ease, and is not distressed even when the noise is loudest. The stridor continues equally loud, although of a somewhat different pitch, when the child is taking the breast. The thorax is tolerably well formed, but there is some forward bulging of the sternum. There is almost no inspiratory indrawing in the episternal notch, but *very much* of the epigastrium and lower antero-lateral parts of the thorax. On auscultation loud snoring is heard all over the chest, and there is marked fremitus.

9th March.—Examined by Dr M'Bride, who reports that "the epiglottis is folded on itself and becomes more so on forced inspiration. At the same time, the air seems to pass through a narrow orifice bounded behind by the arytenoid cartilages and in front and laterally by the folded epiglottis. Cords not seen. Pharynx quite normal."

24th May.—The child is growing fatter and stronger, but is suffering at present from slight bronchitis. The stridor is much less marked and less frequent. It is now heard only during inspiration, and the croaking sound predominates. The noise persists when the nose is pinched, when the tongue is depressed, when the child yawns, and when he is taking the breast. The cough is clear and natural. During screaming, the stridor ceases.

Case V.

Priscilla F., aged 10 weeks, a dispensary patient of Dr George Thyne's, seen with Dr M'Bride at the Royal Infirmary on 25th May 1892. The father is strong and healthy, the mother has frequently suffered from hysteria. The patient is their sixth child. None of the other children ever breathed in the same way. The mother suffered from hysteria during her pregnancy. The labour was

very short and easy. The mother has had little milk in her breasts, and the child seems to have been starved and dyspeptic, but otherwise she has been healthy.

The noisy breathing was not noticed till the end of the first week after birth—when the mother got up for the first time. It seems to be getting louder as the child gets stronger. The sound varies in loudness from time to time, and is often absent for a long time. The mother thinks that it is absent each day for about the same length of time as it is present. When the child is sound asleep she is always quiet; but when the sleep is not profound it may or may not be present.

The patient is a well-formed but rather ill-nourished baby, with no signs of rickets or syphilis or any other disease. The head, heart, lungs, and abdominal organs seem normal. There is no "facial irritability," no cyanosis, no distress, and no movement of the alæ nasi when the breathing is noisy. The fauces, pharynx, and larynx, so far as seen (by Dr M^{c}Bride), are quite normal. Respirations 44; pulse, 144.

The stridor is of the same character as that heard in the other cases, but it is less loud, the croaking part of it is shorter, and it is not nearly so continuous, frequently stopping for quite a long period if the child is undisturbed. Holding the nose, stopping the mouth, depressing the tongue, do not stop the sound. The respirations seem a little quicker when the stridor is present than at other times.

In addition to these more or less severe and typical cases, I have seen two slight ones in otherwise normal children. In these the stridor only occurred for a short time each day, was not very loud, and passed away when the children were a few months old. In one of these cases (which Dr Pirie kindly showed me), after the stridor had almost quite ceased to be heard, the infant had recurrence of the spasm from time to time in a slighter form, accompanied by marked inspiratory indrawing of the chest wall *without* any noise whatever.

I have also seen two apparently similar slight cases in idiot children—both boys—aged 18 months and 3 weeks respectively; but I was not able to be sure in either of them that the noise produced was of exactly similar nature to that in the others.

Clinical Features.

It will be well now to consider separately the various clinical facts observed in the five well-marked cases.

1. *Sex.*—Three of the infants were boys and two girls. This is interesting, because in all the formerly published cases in which the sex is mentioned the patients were girls (9 of Dr Gee's and 5 of Dr Lees'); and the malady is often spoken of as being confined to the female sex.

2. *Family History, etc.*—The parents were all tolerably healthy, but one of the mothers was hysterical and one "delicate." In no case had any other child or relative of the family suffered from a like complaint. In four of the cases the mother was very well during her pregnancy; and also in four of them the labour was very short and easy.

3. *General Health.*—In one case the child was in excellent health and apparently free from dyspepsia. In the other four there was more or less indigestion. In all the cases the intellectual development seemed perfectly good, and there was no abnormality or disease of any organ, except probably a little collapse at the base of the lungs in some of the children. There was no rickets in any of the children when first seen; but it appeared later in those longest observed. "Facial irritability" was absent in the four cases in which it was tested. There was no sign of congenital syphilis in any of the cases.

4. *The Onset of the Stridor* was noticed in one case a fortnight, in another a week after birth; in the rest the noise was observed immediately after birth.

5. *The Course of the Malady.*—Roughly speaking, we may say that in severe cases the stridor goes on increasing in loudness during the first two or three months, and then tends to slow spontaneous improvement. In one case it was said to have disappeared by the time the child was 18 months old. In another it is still present, although much less severe, at 16 months. As improvement goes on, the intervals become longer and the noise less loud; the crowing sound with inspiration is less often heard, only the croaking being usually audible. After the stridor has ceased to be noticed under ordinary circumstances, it may reappear if the child is specially excited or annoyed.

6. *Character of the Abnormal Sound.*—During the presence of the stridor, inspiration begins with a croaking noise, and ends in a high-pitched crow, which two of the mothers correctly described as "very like a hen." When the breathing is quiet, the crow does not occur and only the croaking is heard. Expiration is accompanied by a short croak when the stridor is loud, but at other times it is noiseless.

7. *The Cry* was in one case a little hoarse on one occasion; but as a rule in these cases it is perfectly clear and long and loud. One of the children had a cough, from bronchitis, and it was perfectly normal in character.

8. *The Throat and Larynx.*—In the first case there was some chronic catarrh of the pharynx, and enlarged tonsils; but the larynx, so far as it was seen by Dr Philip and myself, was normal. The other four cases were examined by Dr M'Bride, and I have his authority for saying that the parts were normal so far as they could be seen. In every case the epiglottis could be clearly seen, but the minute appearances of the larynx were concealed by

regurgitated fluid. Although it is quite true that the larynx has occasionally been examined in very young infants, we doubt the possibility of studying in them the mobility of the vocal cords, which requires co-operation on the part of the patient—at least, without the infliction of more pain than the importance of the question at issue seems to justify.

9. *The Inspiratory Indrawing of the Chest-Wall and Episternal Notch* were very slight in Case V., but well marked in the other four cases (especially II. and III.), and when the condition had lasted some time, there was distinct evidence of commencing pigeon-breast.

10. *The Alæ Nasi* moved little, if at all, with respiration, and this was a striking feature in those cases in which the breathing was very laboured.

11. *Absence of Distress or Cyanosis.*—Another most striking feature of the cases is the fact that even when there is extremely marked indrawing of the lower parts of the chest-wall with each inspiration, and the constant stridor suggests the idea of great distress, one finds that the infant appears comfortable and looks about him quite unconcerned. There never was any cyanosis in any of my cases when I saw them.

12. *Intermissions and Variations in Intensity of the Sounds* occurred in all the cases. Even when the condition is most constant and severe, there are from time to time intervals of a minute or more, during which little or no noise is heard, and one often observes an occasional full, long, noiseless breath in the midst of the noisy respirations. Also when the stridor is going on regularly, the loudness of it constantly varies without apparent cause.

13. *Effect on the Sounds of various Influences—*

Mental perturbation always intensifies them, and the intermissions above referred to usually occur when the child is not excited. If, however, the child cries outright, there is no dyspnœa, and the stridor is either quite absent, or at any rate much less marked than when he is merely excited and apprehensive.

Sleep seems sometimes to stop the stridor and sometimes to have no effect on it. Probably the fact is, as two of the mothers put it, that if the child is sound asleep the noise stops, but if he is not sleeping soundly, it goes on as usual.

If the child *yawns*, or his *tongue is depressed* by a spatula, the sound goes on as usual; and it also does so when *the nostrils are closed.*

Taking the breast does not interfere with the stridor. When the mouth is occluded by the nipple, the air entering by the nose is still sufficient to cause a loud noise, although its pitch is somewhat different from that heard during breathing through the mouth.

14. *The Effect of the Ailment on the General Health* is not great, and if, as in Case IV., the child is otherwise quite strong, its growth and development are not interfered with.

15. On the *Effect of Treatment on the Condition* I have nothing to say, except that when, from regulation of the diet or other cause, there was a marked improvement in the child's general health and tone, the local spasm distinctly improved at the same time.

Etiology and Pathology.

With regard to the *proximate cause of the abnormal sounds,* it is interesting to observe that most of the previous writers on the subject seem to have held different views of the mechanism of their production. For example :—

Dr Gee is of opinion that in his cases the sound was produced in the fauces and not in the larynx.

Dr Lees, who had the opportunity of examining a case post-mortem, attributes the respiratory obstruction to a congenital malformation of the upper part of the windpipe, the epiglottis being abnormally "curled inwards," etc.—an exaggeration, apparently, of the normal infantile peculiarities of the parts.

Dr Robertson thinks that the condition is due to paralysis of the postici.

Dr Löri describes with great minuteness the periodic recurrence of a spasmodic closure of the glottis which he found on laryngoscopic examination of his cases.

In my cases, the nature of the sound was such as to render it indisputable that part of it, at least, arose *in the larynx.* Had there been any doubt about this, it would have been dispelled by the fact of the noise persisting, not only when the nostrils were closed, but equally when the mouth was occluded by the nipple, and also when the tongue was depressed by a spatula and during yawning.

It is also, I think, equally certain that the obstruction is due to *spasmodic muscular contraction.* How else can we account for the more or less frequent free intervals, which were so striking a feature of my cases, and which are also noted in all careful clinical descriptions of this ailment ? Or how otherwise can we explain the constant variations in intensity following emotional changes ?

It is interesting to remember in this connexion, that some young infants, who never have any signs of respiratory disorder at other times, make a slight crowing noise with inspiration when greatly excited ; and also that when small babies are coming out of chloroform narcosis they not infrequently make a noise for some time, almost exactly the same as that heard in a marked case of infantile spasm.

As to Dr Lees' case, it seems extremely probable that the local condition which he describes and figures may be the *result* of the constant laboured breathing and not the cause of it. That is to say, that it is an analogous deformity to the "pigeon-breast" which had begun to show itself in those of my cases which had suffered

for a long time, or to the deformity of the nostrils so often seen in patients with obstruction to the passage of air through the nose.

Before proceeding to consider the nature of this spasmodic muscular contraction, it will be well to contrast its main symptoms briefly with those of laryngismus, with which it is often confounded. There are, of course, many obvious points of resemblance between the two conditions, such as that they are both spasmodic respiratory affections, of central origin, occurring in young children. But when we examine further, we find also very essential differences. In fact, we might almost say that the clinical manifestations of infantile respiratory spasm are scarcely more similar to those of laryngismus than the jerkings of chorea are to the muscular contractions of a regular convulsive seizure.

The comparison of a few points will bring this out. Thus :—

1. *Age of Onset.*—Infantile respiratory spasm begins at birth, or within a week or two of it. Laryngismus probably never begins during the first month, and is almost always confined to the period of the first dentition.

2. *Associated Morbid Conditions.*—In infantile spasm we find no rickets and no other associated conditions, except, perhaps, debility and dyspepsia. Laryngismus is almost always associated with rickets, and has very close connexions with tetany, convulsions, and other nervous symptoms.

3. *Character of the Spasm.*—In infantile spasm, each paroxysm is a very small matter, lasting only a second or perhaps two, and causing no distress or cyanosis; but they accompany (in marked cases) a majority of the child's inspirations for a period of months. In laryngismus, on the other hand, each spasm is comparatively very severe and lasts very much longer. They give rise to great distress and cyanosis, but even in the worst cases they only recur at comparatively long intervals.

4. *Effect of other Conditions on the Spasm.*—Infantile spasm is diminished or quite checked by crying; it is not affected by deglutition; and it is often stopped and never aggravated by sleep; while laryngismus is notoriously apt to be set up and aggravated by crying and swallowing, and is especially apt to come on during sleep.

What, then, is the nature of this spasm, and with what other nervous conditions should we class it?

As to its *nature*, we have evidently to do here with a central functional disorder, consisting in a slight disturbance of the co-ordination which takes place in ordinary breathing between the thoracic muscles on the one hand, and those of the larynx on the other. This might be supposed to be due possibly to the presence of some central irritation acting more on the adductors than on the abductors; because, according to Semon and Horsley,[1] there are cortical centres for adduction but none for abduction. It might

[1] M'Bride, *Diseases of the Throat, Nose, and Ear*, 1892, p. 193.

also (and more naturally as it seems to me) be regarded as the result of backward development, and consequent irritable weakness of the central arrangements which control the respiratory movements; just as the tendency to laryngismus in rickety children has been attributed to deficiency of the higher controlling centres (Gay [1]). We would look upon it, therefore, as a "developmental neurosis."

As to its *relationship to other nervous diseases*, it seems to be closely allied to the other "co-ordination neuroses." It has, for example, some features in common with writer's cramp and the various trade-spasms, with the spasmodic strictures of the urethra and other passages, which Sir James Paget [2] calls "stammering with other organs than those of speech," and, especially, with ordinary speech-stammering. To speech-stammering it presents many curiously close analogies. In fact, it bears to the function of respiration very much the same relation as stammering does to that of speech; and "respiratory stammering" would probably be the most suitable name for it, if it had not been already applied to a quite different condition.

Stammering is due to a want of perfect co-ordination between the various muscular mechanisms which work together in the production of speech.[3] Like the other "developmental neuroses," it comes on, as Dr Clouston has pointed out,[4] at a period of life when the function affected by it is attaining, but has not yet *quite* reached, perfect development and stability. Congenital stridor is due, as we have seen, to a similar want of co-ordination; and the function affected by it, although in full use, can scarcely be said to have attained *perfect* development, considering the extreme feebleness of the respiratory movements in new-born infants.

It is also interesting to notice the close resemblance which obtains between some of the clinical features of stammering and some of those of infantile spasm. For example, we have the influence of mental emotion in increasing the difficulty in both ailments, and also the fact that a slight degree of stammering or of respiratory spasm is sometimes set up, in healthy individuals of suitable ages, owing to the influence of mental perturbation. Then, again, we notice the temporary relief which energetic use of the voice affords (singing in the one case, and screaming in the other). Lastly, we have the improvement which, in both cases, follows bettering of the general health.

In conclusion, I wish to acknowledge my indebtedness to Dr M'Bride, who, besides allowing me to make use of one of his cases, and kindly examining most of the infants along with me, has aided me throughout by very helpful suggestions and assistance.

[1] Gay, "On Laryngismus," *Brain*, Jan. 1890.
[2] Paget, *Clinical Lectures and Essays*, Lect. iii.
[3] Wyllie, "On Disorders of Speech," *Edin. Med. Journ.*, Oct. 1891, p. 291.
[4] Clouston, *Neuroses of Development*, p. 9.

Dr M'Bride described a case of this kind which he had seen a good many years ago, and which he assumed to be a disease etiologically similar to laryngismus, and therefore probably a result of cortical irritation. True, there are differences, but there is also a difference between the ages at which the conditions are met with, and Semon and Horsley have shown that the cortical respiratory representation is more marked in young than in more mature animals. Dr M'Bride begged to doubt whether laryngismus ever occurs during sleep, and quoted experiments by Hooper, Horsley, and Semon, on stimulation of the laryngeal nerves during anæsthesia. Dr M'Bride, on the whole, was inclined to think that the disease was similar in etiology to laryngismus, although differing clinically, and exactly like the inspiratory spasm of adults, of which Dr M'Bride had observed several cases.

Dr James Ritchie said that he felt specially interested in Dr Thomson's paper, because although he had seen very few cases resembling those described this evening, one case had caused him some anxiety. He was summoned hurriedly to see a child, age about three months, in consequence of an attack of laryngismus stridulus. Prior to this attack the mother thought that breathing had been quite natural, but for fully a year afterwards there was almost constant laryngeal stridor varying in degree, aggravated by catarrh of chest or digestive organs. Although stridor was very marked, the child seemed to be quite comfortable. Dr Ritchie thought that the pathology of these cases consisted in a hypersensitiveness of the nerve centres along with a certain amount of peripheral irritation, and that as the tensors and adductors of the vocal cords are stronger than the abductors, inspiratory spasm resulted.

Dr James Carmichael had been much interested in Dr Thomson's paper. He regretted very much, however, that the cases on which the paper was based had not been read. He therefore had not the data from which to offer any criticism of them. With reference to the baby just exhibited by Dr Thomson, he took the opportunity of making a cursory examination of it, and concluded that we had to deal with a more or less continuous or tonic spasm of the laryngeal muscles. He observed, however, that while the child was at the breast the spasm apparently ceased, as there was then no stridor audible. The child was evidently of neurotic disposition and tendency, as the mother during her girlhood had been subject to hysteria. The mother also stated that a fortnight ago the child had a fit, which from her description was attended with tonic contraction of the respiratory muscles generally, during which the breathing stopped and the child became more or less livid. He had always felt some difficulty in classifying such cases as those described, differing as they did in some of their clinical features from typical laryngismus. The age of the child and the peculiar nature of the spasm were

elements of distinction in most cases, but he had in his recollection one child during the dentitional period, who was brought to him with typical laryngismus, tonic spasm coming on at irregular intervals with absence of stridulous respiration between the attacks. In this child, however, during the progress of the case the spasm assumed a different nature, being tonic and more or less continuous with stridor, very much as in the baby that had been exhibited to the Society. It seemed that all the physicians who had paid particular attention to these cases were agreed that, pathologically speaking, in typical laryngismus and in laryngeal stridor we had to deal with a similar state, that the disease was essentially neurotic, in its nature depending on functional derangement or instability of the nerve centre. This being the case, he thought we should hesitate before we assumed, from variation in clinical features, that we had a distinct disease to deal with. He was, therefore, of opinion that we were warranted in classing all these cases under the general term laryngismus. As in other nervous affections, similarity, even identity, of pathological state were not inconsistent with variations in clinical features modified probably by the age of the child.

Dr M'Kenzie Johnston said that a condition similar to that described might be occasionally noticed in any healthy infant. It was generally observed after a fit of crying, or some form of emotion, and was probably due to a want of the proper balance of power between the abductors and adductors of the glottis. He wished to ask Dr Thomson if he did not think that this was a temporary form of the more permanent condition met with in his cases. It was difficult to account for the persistence of the affection, and he doubted whether it could be produced by any local cause.

Dr Thomson thanked the members of the Society for their reception of his paper. He did not agree with Dr M'Bride and Dr James Ritchie in thinking that the recurrent spasm was necessarily produced by the presence of some cortical irritation, but thought it much more likely that the want of co-ordination was to be explained by supposing that the higher controlling centres were backward in their development, and that this interfered with their having full control of the lower reflex ones. He also did not think that the mere difference in age could account at all for the difference between the symptoms of the cases under consideration and those of laryngismus. He had seen lately a case of real laryngismus in a very young infant, and the symptoms were just like those in an older child. He disagreed with the inferences which Dr Carmichael had drawn from some statements which the mother of the infant shown had made, and thought that, on more careful inquiry, it would be found that the "fits" the child was said to have had were really only manifestations of acute indigestion from injudicious feeding. He quite agreed with Dr M'Kenzie Johnston that healthy children

often made a crowing sound with inspiration when excited, very like that of the condition described,—just as we were accustomed to hear some adults stammer when they were excited, who never did so at any other time.

3. TWO HUNDRED AND FIFTY-EIGHT CASES OF SUFFOCATION OF INFANTS.

By Charles Templeman, M.D., B.Sc. (Pub. Health), Surgeon of Police; Surgeon to the Royal Infirmary, Dundee.

In every town which contains a large proportion of the industrial classes cases of overlaying are more or less common.

In the city of Dundee, during the ten years from 1882 to 1891, no fewer than 399 cases were reported to the police in which infants had been found dead whilst in bed with their parents. Of these I have personally examined 258 children who have died in this way from overlaying. The large number is accounted for to a great extent by the nature of the population, which is mainly industrial. The staple industries are the spinning and weaving of jute and flax. A very large number of women are employed in the mills and factories, and many of them are married and have children.

The population according to the census of 1891 was 153,587. Of these above one-sixth live in houses of one room, and above two-thirds of the inhabitants occupy houses of either one or two rooms. Cases of overlaying are almost entirely confined to this class, and about three-fourths of the cases seen by me occurred in houses of one room. In a large number of instances the parents were of dissipated and dissolute habits, living amidst squalor and filth, with often little or no furniture in the house, which was sometimes even devoid of a bed. The following table shows for each of the years,—the number of cases examined; the number of illegitimate children amongst them, with the proportion of these to the total illegitimate births during the same year; the number of deaths which took place between Saturday night and Sunday morning; the number which occurred during the winter months, from October to March, and also the number which occurred in each month of life.

It will be observed that in all these cases the child was under the age of nine months. The risk of a child being suffocated whilst in bed with its parents is of course greatest in the earliest months of life. As it gets older it is better able to struggle and avert this calamity, and after it has reached the age of nine months the risk of death from this cause seems to be practically *nil*. These statistics show that the liability of an infant to this form of death remains constant during the first three months of its existence, and after that period it rapidly diminishes in every succeeding month.

Year.	No. of Cases.	Illegitimate.	Cases occurring between Saturday night and Sunday morning.	Cases occurring from October to March.
1882	19	7=14·1 per 1000	10	12
1883	26	7=13·3 „ „	16	14
1884	24	8=14·9 „ „	10	11
1885	24	9=16·4 „ „	12	18
1886	28	10=20 „ „	11	22
1887	32	13=26·3 „ „	12	23
1888	28	10=20·9 „ „	13	17
1889	17	3= 6 „ „	10	11
1890	34	9=18·5 „ „	13	19
1891	26	7=15 „ „	11	12
Total	258	83=32 % of the whole.	118=46 %	159=62 %

Of these there occurred in the

1st month.	2nd month.	3rd month.	4th month.	5th month.	6th month.	7th month.	8th month.	9th month.
62	67	66	21	16	13	5	4	4

In cases of overlaying suffocation is produced in one of three ways—

1. By the infant's face being firmly pressed against the mother's breast, thus mechanically obstructing the entrance of air to the lungs.

2. By the bedclothes being firmly placed over the child's face, with a similar result.

3. When the child is placed between its parents, or by the side of one of them, who while asleep turns over upon it, thus causing death by overlaying proper.

The usual history obtained is as follows:—The child is put to bed in its usual health. When the mother retires, or at some other time during the night, she places the child on one of her arms, and puts it to the breast. At that time nothing unusual is observed. The mother falls asleep with her infant still at the breast and resting on her arm, and in the morning when she awakes she finds it in this position dead.

The external appearances presented by the body are chiefly of a negative character. There are no marks of violence to be observed. As a rule there is no flattening of the nose and face from pressure. Post-mortem lividity comes on early, and is specially well marked on that side of the body on which the infant has been lying; the face is placid and calm; the eyes sometimes slightly congested, but not staring; the lips are livid, and the tongue not protruded. Frothy mucus, often tinged with blood, is generally seen about the mouth and nostrils. The hands are sometimes tightly clenched.

Unfortunately in the majority of these cases I have not been permitted to make a post-mortem examination, but those bodies on which I have had an opportunity of making a complete or partial examination presented the usual appearances found in

cases of death by asphyxia, viz., a varying degree of congestion of the cerebral membranes—more or less engorgement of the internal organs, especially the lungs and kidneys, and the large thoracic veins, a fluid condition of the blood, which was dark in colour; and generally a distended condition of the right side of the heart, while the left was nearly or altogether empty and contracted (in one case both sides of the heart were completely empty). In about half the cases examined small punctiform hæmorrhages were observed beneath the pleura and pericardium. The larynx, trachea, and bronchi were, as a rule, congested, and contained some frothy, often blood-stained mucus.

The principal causes producing this great mortality from overlaying are,—

1. Ignorance and carelessness of mothers; 2, drunkenness; 3, overcrowding; and, 4, according to some observers, illegitimacy and the insurance of infants.

In many cases parents are not aware of the danger of having their infants sleeping along with them and resting on their arms, and on this account overlaying frequently occurs in a perfectly accidental manner. In other cases, however, parents are quite aware of this danger, but either from carelessness, for convenience to themselves, or from an utter disregard for the child's life, they wilfully allow it to run the risk of being suffocated. Thus I have known several cases in which women have lost more than one child from overlaying.

It has long been notorious that a very large proportion of these deaths occurred between Saturday night and Sunday morning, and early in my official career I was struck by the frequency with which I was called by the police on Sunday mornings to examine the bodies of infants found dead in bed beside their mothers. The fact that no fewer than 118, or 46 per cent., of my cases occurred at this particular time shows that the coincidence is more than an accidental one. The only explanation of this is no doubt that, receiving their week's wages on Saturday, many of the lower classes, among whom these cases are so common, indulge freely in drink, and go to bed more or less intoxicated. Although the parents are generally unwilling to admit this, the evidence of their neighbours is often sufficiently clear to establish the fact.

It has also been suggested as a possible explanation of this striking fact, that on Saturday nights the parents going to bed at a much later hour than usual, more fatigued than on other nights, and with the knowledge that they do not require to rise to their work early in the morning, sleep more soundly than usual, and thus increase the risk of causing the death of their children in this manner. This may have some slight bearing on the point, but from my experience I have no hesitation in affirming that the question of drink is a much more important one, and plays a very prominent part in producing this frightful mortality.

Another important cause is, however, overcrowding. As I have already said, most of these cases occur in one-roomed houses, where frequently father, mother, and two, three, or even as many as five children sleep in one bed, which sometimes consists of a few jute sacks spread out on the floor. Lying on these, with very scanty covering, they huddle together to keep themselves warm, and in this way the suffocation of an infant, so easily accomplished, is not difficult to explain. This helps to account for the fact that 159, or 62 per cent., of my cases occurred during the winter months.

The large number of illegitimate children who meet their death in this way has frequently been commented upon. That 83, or 32 per cent., of my cases belonged to this class is no doubt a striking fact, considering that during the same period the birth-rate of these unfortunates averaged 10·3 per cent. of the total births in our city. But this is easily explained when we consider that these cases are entirely confined to the lowest class of the workers in our mills and factories, who are usually poorly fed, badly clothed, often of dissolute habits, and live in miserable lodgings, while the children themselves are, as a rule, improperly fed and more or less neglected. As a matter of fact, I have been unable to detect any circumstances which give rise to stronger suspicions of criminal culpability in these cases than in others of a similar nature occurring in children of a different class.

The question of the insurance of children and its effect on infant mortality is one which has lately been receiving a good deal of attention, and some observers are of opinion that it has not a little to do with the frequency of overlaying. In the report of the Select Committee of the House of Lords on the Children's Life Insurance Bill of 1890, there is much valuable and interesting information on this point. In the opinion of many of the witnesses examined, especially medical men and coroners, the paltry sum for which their children have been insured has proved too great a temptation to some parents, who have by neglect, if not by more direct and speedy measures, sacrificed their children's lives.

On account of the ease with which overlaying can be accomplished, and the certainty that any criminal intention will escape detection and punishment, it is believed that some parents of the lowest class, who are unwilling to undertake the support and upbringing of their children, are induced to take this means of ridding themselves of what they feel to be a burden, and the small sum of money for which the infant is insured to some society or burial club is considered an additional inducement for them to commit this crime.

In 100 of my cases I made careful inquiries into the question of insurance, with the following result:—In 46 cases no insurance whatever had been effected on the child's life. In the case of 26 children who were insured their parents were not entitled to

benefit on account of the policy not having been in force for three months. Under such circumstances some societies occasionally give a gratuity to the parents, but they invariably decline to do so in cases of overlaying. The rest of the cases, 28 in number, were either partially or fully insured, and the parents entitled to sums varying from 7s. 6d. to 45s. In 16 of these 100 cases the child had to be interred at the expense of the Local Authority.

As a rule, amongst that class in which overlaying is most common, only one insurance is effected on the infant's life. The usual premium paid is one penny per week, and from time to time, as the family increases, the insurance is increased to include them all. Many offices do not insure children at all till they are a fortnight old, and the policy must be in force for three months before the parents can derive any pecuniary benefit from the child's death. In other cases, again, the child must be insured for twelve months before the parents are entitled to full benefit. As a rule the maximum amount payable at the age of nine months, when overlaying practically ceases to occur, is from 45s. to 50s., although this is occasionally increased by the parents being members of some local friendly society entitling them to £1 or 30s. on the death of one of their children.

As regards illegitimate children, the majority of insurance offices will not accept them till they have reached the age of three years. It will thus be seen that the amount obtained by the death of an infant at this age is but small, and in most cases insufficient to cover the expenses of a funeral conducted in the plainest possible manner.

It is, of course, open to question whether it is advisable to allow parents to have a money interest on the death of their children, but in these cases the amount obtainable is so insignificant that I do not think that alone would induce any parent to commit the crime of child murder. As a matter of fact, as a result of my investigations I have not been able to see any grounds for believing that this high infantile mortality has any connexion in a criminal sense with the question of insurance.

Can nothing be done to arrest this serious leakage of infant life? There is no doubt that deaths from overlaying are distinctly preventable, and such being the case, the responsibility for its occurrence ought to be fixed on some one. When, however, we come to inquire into the degree of culpability to be attached to the parents, we at once meet with a difficulty. The physical appearances, both external and internal, give us no clue in determining whether the death has been accidental or homicidal. The suffocation of an infant without any marks of violence is such an easy matter, that a medical examination alone is insufficient to bring home culpability to the parents. The ease with which this can be accomplished is well illustrated by the two following cases:—

In the first case, the child was four months old, well nourished, and of healthy appearance. The mother along with her infant had been spending the day in the country, and on returning at night had been driven to within 300 yards of her own door. Before leaving the conveyance, she put her infant to the breast, and wrapped a shawl around it, placing it over its head, as the night was somewhat chilly. The woman was perfectly sober. On arriving at her house she was horrified to find her infant dead.

In the other case, a mature, healthy child, 7 days old, was put to the breast by its mother, who was sitting up in bed and carrying on a somewhat animated conversation with some friends who had called to see her. She was a stout woman, with large, soft, and flabby breasts. She was paying little attention to her infant, and on withdrawing it from the breast, about five minutes afterwards, found it was dead. Both these cases presented the usual appearances of death from suffocation, and this had taken place while the parents were awake, and they were quite unaware of anything unusual going on.

The number of deaths from this cause is such an important factor in our infant mortality that any proposition calculated to lead to their diminution deserves consideration.

To my mind the only safeguard to its occurrence is that the child should be made to occupy a separate cot from its parents or nurse. No doubt with due care a child may be allowed to sleep with its mother without any harm resulting; still the large number of these cases which occur without any apparent wilful carelessness on the part of the mother shows that nothing short of this measure being made compulsory will insure absolute safety to the child. In my opinion, although perhaps savouring somewhat of grandmotherly legislation, we ought to adopt a modification of the German law, which prohibits a child under the age of two years from being allowed to occupy the same bed as its parent or nurse. No doubt such an enactment in the case of the poorest classes might be difficult to enforce, and would entail some hardship and considerable inconvenience, still it would have this salutary effect, that in the case of an infant being overlaid the responsibility for the occurrence would be fixed on the parents.

At present the impossibility of obtaining evidence of direct culpability permits all such cases to be passed over with impunity. However strong the suspicion of criminal culpability may be, and in some cases the conduct of the parents has given grounds for raising grave doubts as to their innocence, it is almost impossible to get evidence of a kind which would be sufficient to obtain a verdict from a jury. This is shown by the fact that in none of these cases did the Crown authorities deem it advisable to order a prosecution.

But even short of this measure, the punishment of parents who

can be proved to have gone to bed in a state of intoxication would have a beneficial effect.

We have already our Protection of Infants' Act, which provides that "any person over sixteen years of age, who having the custody, control, or charge of a child, being a boy under the age of fourteen years, or being a girl under the age of sixteen years, wilfully ill-treats, neglects, abandons, or exposes such child in a manner likely to cause such child unnecessary suffering or injury to its health, shall be guilty of a misdemeanour, and on conviction thereof on indictment shall be liable to a fine not exceeding one hundred pounds; or alternatively, or in default of payment of such fine, or in addition to payment thereof, to imprisonment with or without hard labour for any term not exceeding two years." I do not know if this Act could be considered applicable to such cases as I have mentioned, but if not, it should certainly be made to embrace them, for without doubt such drunken parents "wilfully expose their children to unnecessary suffering," and endanger their lives, and ought to be made directly responsible for their death from such a cause as overlaying.

One case to illustrate this. One afternoon, in April 1890, an infant five weeks old was found dead in bed in its mother's arms. The mother had been drinking during the forenoon, and had gone to bed intoxicated about half-past one o'clock, taking her infant with her. Shortly after two o'clock she was seen lying in bed on her right side with her child resting on her right arm, and its face turned towards her right breast. As the child was crying, the mother was awakened, and asked to put her infant to the breast. She did so, and immediately fell asleep. About four o'clock her husband returned, and found her lying in bed in the same position, with her breast bare, and the child close to it. The child was dead, but the mother was not lying upon it. There was some bloody, frothy mucus about its mouth and nostrils. A post-mortem examination showed that the immediate cause of death had been asphyxia.

Now, if the law is powerless to reach such a clear and flagrant case as this, it surely stands in urgent need of amendment, and in my opinion it ought to be amended on lines such as I have already indicated.

Dr Henry D. Littlejohn complimented Dr Templeman on his paper, and agreed with him as to the continued prevalence of this form of infantile mortality. While the authorities might, in certain districts of the country, be open to the imputation of slackness in dealing with cases, it must be remembered that too often the evidence, even where a post-mortem examination of the deceased is granted, is imperfect, and the medical man finds himself unable to speak with decision as to the exact cause of death. So far as his experience went in Edinburgh and neighbourhood, the Crown

always ordered a prosecution—*first*, where a mother while under the influence of liquor went deliberately to bed with her infant after being duly warned as to the risk of so doing; and, *second*, where the body of the deceased showed marked evidence of pressure. The excellent statement of Dr Templeman would undoubtedly have the salutary effect of drawing public and official attention to this increasing source of mortality in the young.

Dr Templeman thanked the Society for the favourable manner in which his paper had been received. In reply to Dr Littlejohn's criticism with regard to the action of the local officials, all he could say was that they acted in accordance with instructions received from the Crown authorities. What he desired to emphasize in his paper was, that if the law was powerless to cope with such cases as some of those described, it should be amended so as to embrace them.

Meeting XI. (Special)—June 23, 1892.

Mr Joseph Bell, *President, in the Chair.*

Original Communication.

TRANSFUSION OF HUMAN BLOOD IN THE TREATMENT OF PERNICIOUS ANÆMIA.

By David J. Brakenridge, M.D., F.R.C.P.Ed., Senior Physician, Edinburgh Royal Infirmary; Lecturer on Clinical Medicine, Edinburgh School of Medicine, etc.

Mr President and Gentlemen,—I do not intend in this paper to attempt any serious discussion as to the pathology of pernicious anæmia, nor shall I spend time in endeavouring to *prove* that all the cases which I shall bring forward were incontestable examples of that disease. I *believe*, however, that the following five cases—in which nine transfusions have been performed—were cases of pernicious anæmia.

They certainly possessed the following claims to be included in that group:—In all of them the type of the anæmia was that met with in pernicious anæmia: it could be traced to no sufficient cause, and in spite of the most improved ordinary remedial measures—including drugs, dieting, and general management—it tended towards a fatal result.

In this paper—long as it is—I am obliged to omit, at present, a large number of the observations made and recorded in my reports, and to limit myself to the evidence afforded by the blood.

Before proceeding to discuss the influence of transfusion on pernicious anæmia it is absolutely necessary to glance in the

briefest manner at the facts known and the views entertained regarding this disease. The following facts are recognised on all hands:—

First, The disease is characterised by a progressive and remarkable reduction in the number of the red blood corpuscles; they may fall to a fifth or a tenth of the normal percentage. This reduction is accompanied by various alterations in form, size, and characters.

Second, Along with this, there are evidences of a greatly increased and peculiarly modified destruction of blood corpuscles in certain organs, such as the liver, one of the normal functions of which is to destroy exhausted blood corpuscles which have served their purpose and lived their life.

Third, There are evidences of altered genesis of blood corpuscles in the blood-forming organs, such as the marrow of bone, &c.; that most usually observed being an over production in the number, and an under production in the quality of the corpuscles.

Fourth, These abnormal conditions are not definitely attributable to any ascertainable cause, and differ in essential characteristics from those observed in all other anæmias.

Theoretically, such an anæmia, with all these coincident facts, might be accounted for in a variety of ways:—

1*st*, *A faulty genesis of corpuscles in the blood-forming organs*,—the result being corpuscles which are imperfect, deformed, delicate, short-lived, and easily destroyed by any destroying agent, *e.g.*, by the action of the ordinary blood-destroying organs, such as the liver, or by any poison.

2*nd*, *An over activity, and perhaps a modified activity, in the organs which normally destroy the old worn-out blood corpuscles:* the changes in the blood-forming organs and in the corpuscles being merely secondary.

3*rd*, It is quite possible that *both of these factors might be coincident:* imperfect formation and excessive destruction of the blood corpuscle being both results of the same disturbing cause.

4*th*, The starting point of the whole process might be *some poison—ptomaine or other*—introduced through the alimentary canal or otherwise.

5*th*, It should be added that, while the most characteristic cases of this disease have arisen independently of any other discoverable morbid condition, there is every reason to believe that the group of facts and phenomena which are characteristic of this disease—pernicious anæmia—does occasionally *become engrafted upon a wide variety of pre-existing diseases—e.g.*, chlorosis, tape worm, and many others. It is *none the less* a *new disease* on that account, but its clinical features are thereby modified. The pre-existing disease is probably distinctly predisposing.

Many mines of information in the field of the pathology of the disease have recently been opened up, and worked with no little

success by such pathologists as Dr Russell of our own school; my former pupil Dr William Hunter, Dr Mott, Rindfleisch, and a host of others. Much precious material has been brought to the surface; but I think it will be admitted that, though *some links* have been successfully forged, a *chain of evidence which will bind all the facts into one consistent theory has not yet been put together.*

Indeed, the pathology of this remarkable disease is still most obscure, and the information chaotic. I therefore offer the following cases as a humble contribution, in the hope that it may add something, however small, to the elucidation of this subject.

Now, there can be no doubt that the view entertained, *à priori,* regarding the *probable nature* of the disease, will very materially influence the *expectation of success* from such a remedy as transfusion of human blood. Thus, if we believe the explanation of the anæmia is to be found in a vicious activity of the blood-destroying organs, and particularly if we hold, with Hunter and others, that this is caused and aided by a blood-destroying poison absorbed from the alimentary canal, we could expect very little and very temporary assistance from the introduction of so small an amount of blood as from 2 to 6 ounces.

On the other hand, if we believe the explanation of the anæmia to be this:—That, owing to some modification of their function, the blood-forming glandular organs have lost their power to produce healthy, properly constituted, and normally long-lived blood corpuscles, our expectation of a beneficial result should be very much more hopeful. For in the former case the blood corpuscles added by transfusion, and considerably injured by exposure during the operation might be expected to be destroyed in their first circulation through the portal system. In the latter case, however, we might anticipate that the addition of even so inconsiderable an amount of rich healthy blood, containing normal blood corpuscles, would improve the condition both of the faulty blood-forming organs and of the faulty blood with which it came into contact. It was in the hope inspired by the latter view—which I held in 1885—and which I still hold with some modifications, that I determined to give transfusion a fair trial in all my cases of pernicious anæmia which failed to respond to other rational and approved methods of treatment. From the outset I believed that, to estimate the value of transfusion fairly, it would be necessary, should the first transfusion fail to accomplish a cure, to repeat the operation as often, and at such intervals, as the progress of the case should demand. No one would judge the value of the most powerful remedy from the administration of a single dose.

I have excluded from this paper four cases of pernicious anæmia, more or less advanced, which responded satisfactorily to ordinary treatment, more especially to arsenic in carefully regulated doses.

The following group comprises only those in which, all other rational treatment having proved useless, transfusion of human

blood was resorted to as a *dernier ressort*, but as a last resource which seemed from the first to promise the very best results. I offer them for your consideration, then, in the belief that they in some measure support the following conclusions :—

1. That in the transfusion of human blood we have a powerful means of arresting the progress of this grave disease.

2. That the operation in skilful hands, and with due precautions, is perfectly safe.

3. That a number of precautions must be attended to in performing transfusion, if inconvenience and even risk to the patient are to be avoided.

4. That the results observed in these cases throw a suggestive side-light on the probable nature of the disease, supporting, although not absolutely shutting one up to the view that the anæmia is probably mainly due to a failure in blood genesis rather than to an excess in blood destruction.

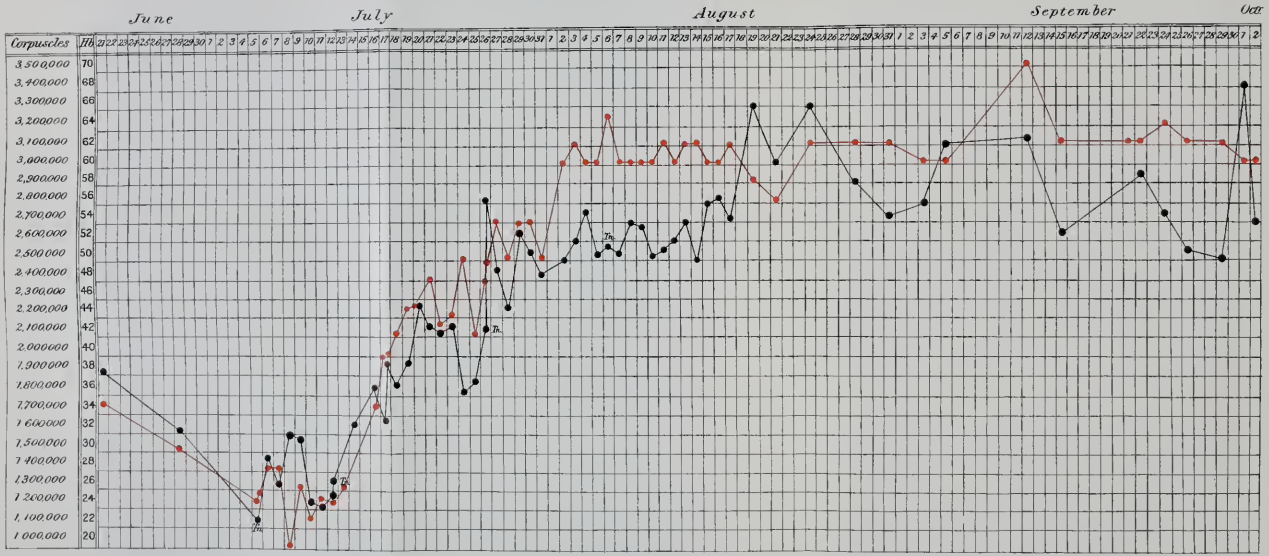
While, as I have stated, I believe that the results of transfusion must be taken into account in any theory of the pathology of the disease, I may now say that the purpose of this paper is *therapeutical* rather than *pathological*, and therefore I shall not waste time over any further discussion of the unity or diversity of the forms of pernicious anæmia, but will found my diagnosis of the cases dealt with in this paper on somewhat broad lines ; for the physician cannot afford to wait until pathologists have settled all their nice points of difference regarding diseases before he treats those suffering from them. Indeed, of many diseases it may be said that the treatment is nearly perfect, while the pathology still remains as mysterious as ever.

The following are the cases very much abbreviated :—

Case I.—Mrs G., married, æt. 34, housewife, came of a fairly healthy stock. Between the ages of 16 and 22 she was in New Jersey, and while there she suffered from ague. She, however, was quite well after her return to this country, and was married when 24 years of age. She had four children ; and although in connexion with the births of some of these she suffered considerably, she had intervals of good health. For two years before the birth of her fourth child she was very well. After this child was born she fell off and became very weak, and there was at this time some swelling of the glands of the neck. This, however, disappeared under treatment. She was never quite strong after this. In the summer of 1884 she again became pregnant, and during her *tempus pariendi* she was much troubled with sickness and purging.

On February 27, 1885, she gave birth to a seven months' fœtus. After this she was very weak and torpid for a month, then she improved somewhat for a time ; but again she fell off, and grew weaker and weaker, until she was admitted into the Infirmary under my care on 19th June 1885. She had been a good deal

Case 1. Mrs. G.



The black line indicates the blood corpuscles; the red line the hæmoglobin.

troubled with dyspepsia before she came in. For her weakness, quinine and iron had been administered without avail.

On admission her weight was only 6 st. 3 lbs. She was deadly pale, and her face and body generally had a yellowish tinge; but there was no jaundice, dropsy, or cyanosis. There were a few swollen glands on the right side of the neck; none anywhere else. The spleen and thyroid were normal in size. Examination of the blood showed the following conditions:—Hæmoglobin, 35 per cent.; red blood corpuscles, 1,900,000 per cmm.; tailed corpuscles, 5 per 100; leucocytes, 5 per 100 squares. There were numerous microcytes. The red blood corpuscles were extremely unhealthy looking, and varied in size, many of them being much smaller than normal.

Examination of the circulatory organs elicited the ordinary evidence of the results of extreme anæmia, viz., faintness, occasional palpitation, systolic murmurs in all the areas, a *bruit de diable* in the deep jugular veins, and a weak, compressible, but regular pulse, about 80 per minute. She suffered from flatulence, heartburn, and acidity after meals. The bowels were rather costive.

The eyes were examined on July 10th, and the remains of an old hæmorrhage was seen in the retina in the left eye alongside a vessel running upwards and outwards from the fundus. She suffered from amenorrhœa. All the other systems were fairly normal.

She was admitted, as I have said, on the 19th of June 1885, and on that day, owing to extreme weakness and rather troublesome dyspepsia, she was put on milk diet. This was, however, after a few days changed to convalescent diet. The blood was examined for the first time on the 21st of June, two days after admission, and the characters were found to be as above stated.

On *June 27th* she was ordered, in addition to other food, one peptonized beef suppository every four hours by the bowel, and a mixture containing 5 grs. of citrate of quinine and iron and half a minim of Fowler's solution of arsenic.

On *June 28th*, a week after the first examination, the hæmoglobin of the blood was found to have fallen to 30 per cent., and the red corpuscles to 1,600,000 per cmm.

On *July 3rd* the quinine, iron, and arsenic mixture was discontinued, as it was disagreeing with her stomach. Liquor arsenicalis in ℥ij. doses was ordered three times daily immediately after food.

On *July 5th*, a week after the last examination of the blood reported above, it was found that the hæmoglobin had fallen to 24 per cent., and the red blood corpuscles to 1,160,000 per cmm.

The temperature from the first was above normal, ranging irregularly between 99° and 100·8° F.

The patient was becoming much weaker, and as the red blood corpuscles had fallen in a fortnight from 1,900,000 per cmm. to 1,160,000, and the hæmoglobin from 35 per cent. to 24 per cent.,

it was clear that there was a steady progressive deterioration of the blood for which no sufficient cause could be found. This deterioration continued to progress in spite of arsenic and careful dieting.

The diagnosis in this case lay between lymphadenoma and pernicious anæmia. The characters of the blood, and the great reduction in number of the red corpuscles, however, and the absolutely stationary and localized condition of the enlarged glands in the neck, rendered it almost certain that—however it might have originated—*the disease which we now had to deal with was pernicious anæmia.*

It is very exceptional to find so severe an oligocythæmia—apart from any hæmorrhage—in any of the forms of wasting disease, such as carcinoma, lymphadenoma, or unless in very rare cases in which it is probable that a true pernicious anæmia has become superadded. Indeed, in very few wasting diseases is the number of the red blood corpuscles per cmm. reduced—even in the *extremest state* of *anæmia* caused by them—below 3,000,000 or 2,000,000 per cmm. The unanimous opinion of a large number of physicians, who saw the case at the time, was that it must be regarded as a case of true pernicious anæmia. The question now arose, Could anything further be done to arrest the progress of the disease in this patient? Arsenic was ill borne in even ℥ij. doses, and it with careful nursing, and dieting by stomach and bowel, had failed to arrest the downward progress.

At this time—seven years ago—Dr William Hunter had not published any of those able researches which have since given so strong an impetus to the view that the disease is a hæmolytic rather than a cytogenic disease, *i.e.*, is due rather to excessive destruction of blood corpuscles than to their faulty formation and consequent tendency to early death.

I held, as I am still disposed to hold, that pernicious anæmia is mainly due to a *faulty genesis of the corpuscles in the blood-forming organs, and a consequent tendency to their early death in the blood-destroying organs.* That the disease was not necessarily incurable was shown by the beneficial action of arsenic in many cases of pernicious anæmia. I determined, therefore, to try the effect of *repeated transfusions*, in the hope that the normal function of the blood-forming organs might thereby be restored; or that, even should transfusion, performed at intervals, not suffice alone to cure the disease, its occasional performance from time to time might *give such an impetus to* the *blood-forming organs* as would bring them within the influence of the curative action of arsenic.

At this time transfusion of human blood was in very bad odour as a remedial measure—being regarded as both dangerous and useless by most authorities. I asked Dr John Duncan to see the case with me. Indeed, this is the case to which, as some of you may remember, Dr Duncan referred in 1886 in his able paper "On

Reinfusion of Blood in Primary and other Amputations," as having suggested to him the idea of transfusion in surgical hæmorrhage. It has thus an additional and historical interest. After full and careful consideration of the matter, and of the various methods of transfusion, he advised the indirect method, and agreed, as soon as I should think fit, to operate. On July 5th I advised the operation, and Dr Duncan performed it at 12 noon.

The blood was taken from a very strong, healthy student, Mr Thomas White. It was about the average in richness in blood corpuscles, *i.e.*, 5,000,000 per cmm. I need not detail the operation, as it has already been given by Dr Duncan in the paper I have mentioned, and which is published in the *Transactions* of this Society for 1885-6. Suffice it to say that the indirect method was employed, and the blood was kept fluid by admixture with $\frac{1}{3}$ part of its bulk of a 1 in 20 solution of phosphate of soda in distilled water kept at blood heat. Six ounces of the blood with two ounces of the phosphate of soda solution in all were injected. During the operation I kept my finger on the patient's pulse, and we regulated in this way the rate at which the injection was performed by Dr Duncan, who slowed the proceeding when any marked change in the pulse-rate or respirations was noted. As the transfusion proceeded it was observed that the pulse steadily increased in force and volume, but did not alter in rate.

At 7 P.M. the same evening, it is stated in the report,—"Patient feels better; her pulse has decidedly *increased in volume.* After the transfusion she had a motion of the bowels which was slightly loose." Seven hours after the transfusion the blood was again examined, and the red corpuscles were found to have risen from 1,160,000 to 1,470,000 per cmm., *i e.*, there was an increase of 310,000 per cmm. Curiously this corresponded almost exactly with an approximate estimation made before the transfusion by calculating the probable total amount of blood in the body, and the proportional amount added by injecting 6 oz. of blood. On this, as on all other occasions, before and after the transfusions, the observations were checked, a large number of estimations being made and an average taken.

The next point which we were anxions to note was this:—Would this addition to the blood corpuscles be *rapidly lost?* or would evidence of its *beneficial action on the blood formation* be shown by a *permanent and progressive improvement* in the blood?

Neither Dr Duncan nor I expected from this first transfusion more than a comparatively transient effect. My determination, however, was that whenever a decided declension in the number of the blood corpuscles should once more become apparent, we should again transfuse without delay. As a matter of fact we let the patient down nearly as far as she had been previous to the operation, before it was repeated. But before we pass on, let us glance at the main effects of this first transfusion. These were as follows:—

First, There was not only an immediate gain in the number of corpuscles per cmm., but for a short time thereafter there was a slight upward tendency in both corpuscles and hæmoglobin. This was maintained for about three days, and then there was a rapid declension until five days after transfusion, when almost as low figures were reached as before the operation had been performed.

Second, Another marked alteration in the blood is noted in the report as follows:—"Each examination of the blood before the transfusion showed numerous microcytes: the red blood corpuscles were extremely unhealthy looking, many of them being much smaller than usual; and the leucocytes numbered only 5 in 100 squares. On examining the blood after transfusion, it was found that the microcytes had gradually diminished in number, and the red blood corpuscles had assumed a more healthy appearance. About five or six days after the first transfusion, no microcytes were visible; and they were never seen at any subsequent examination. The red blood corpuscles had also greatly improved in size, and regained their normal appearance: this healthy appearance they maintained until patient left the Infirmary nearly cured."

Third, After the second transfusion the temperature, which before the transfusions were commenced had ranged from 99° to 100·8°, gradually fell, and on August 30th became normal. It remained so until she left the Hospital.

Fourth, Patient's general condition decidedly improved. The pulse continued to be fuller and firmer. The cheeks and lips were better coloured, and patient felt altogether better. This general improvement continued to advance progressively until the patient left the Infirmary; *but I am anticipating*, as we had still to repeat the operation on three subsequent occasions before she left.

The second transfusion was performed on July 12th.

The corpuscles before transfusion numbered		1,300,000	per cmm.
„	after „ „	1,670,000	„
	Increase,	370,000	„

The third transfusion was performed on July 26th, while the corpuscles were rising in number.

The corpuscles before operation numbered		2,215,000	per cmm.
„	after „ „	2,820,000	„
	Increase,	605,000	„

Part of this increase may be attributed to *an improved resistance to destruction on the part of the corpuscles from admixture with healthy blood.*

The fourth transfusion, performed on August 7, was unsatisfactory in this respect, that a 10 per cent. instead of a 5 per cent. solution of phosphate of soda was employed. Now the action of too strong a solution of that salt is to damage the blood corpuscles.

There was, therefore, not the usual rise after this operation, but the reverse.

The corpuscles before transfusion were	2,575,000	per cmm.
„ after „ „	2,575,000	„
Decrease,	70,000	„

She refused to be transfused any more, although strongly advised to have the operation repeated until she was quite restored. When she left the Hospital, on October 2, her blood corpuscles were about 3,000,000 per cmm., and her hæmoglobin 60 per cent., both having been trebled since the first transfusion. Her strength and colour had greatly improved. I did not consider the cure complete, although I have no doubt that she would have been cured had the treatment been continued.

For some months she remained fairly well, and able for her household duties. The anæmia, however, recurred, and although the treatment by arsenic was again prescribed by the late Dr White, her physician, she died a few months after her return to Yetholm of the same disease.

Case II.—Mrs A. M., æt. 25, housewife, married, born at Dalgetty, residing at Crossgates, Fife, was admitted into Ward XXXIII. of the Edinburgh Royal Infirmary on October 11, 1888.

Her family history was remarkably good. Her father and mother, as well as her two children, were all alive and very healthy. About 7½ months before admission her present illness commenced without any definite cause. Her appetite began to fail, she lost flesh considerably, and sometimes she was sick after meals. These symptoms gradually increased, and she became so weak that she was advised, and decided to come into the Infirmary. No other symptoms had developed. Patient had not menstruated for eight months. This was all the history.

The following is a summary of her state on and after admission:—Height, unascertained; weight, 6 st. 7½ lbs.; muscularity, very poor. Her face was fairly well filled out: by no means emaciated, but extremely pale. The conjunctivæ were almost absolutely white; the lips and gums were also very colourless. The skin had a distinctly lemon-yellow tinge. There was no cyanosis, jaundice, or dropsy. There was no swelling of the lymphatic glands, or enlargement of spleen or liver. On admission the appetite was deficient, and she suffered from indigestion, which improved greatly shortly after she had been put on peptonised light food. Examination of the blood and circulatory system showed considerable deviations from the normal.

The following marked and characteristic alterations in the blood were present:—

First, A pronounced oligocythæmia, *i.e.*, the red corpuscles were

greatly reduced in number. On admission, October 11, instead of the normal number, 5,000,000 per cmm., there were only 1,000,000. Later on they fell so low as 640,000 per cmm.

Second, There was what Quincke has termed "poikilocytosis," *i.e.*, a remarkable variation in the form and size of the red corpuscles. Some were tailed, some notched, some larger and some smaller than normal, and so forth.

Third, There were numerous microcytes—small coloured corpuscles not usually met with in blood—and which Eichorst believes to be pathogenic of pernicious anæmia.

Fourth, There were a number of distinctly nucleated large red blood corpuscles.

Fifth, There was no increase in the number of the white corpuscles.

Sixth, There was a slight increase in the blood-plates.

Seventh, The hæmoglobin was as low as 20 per cent.

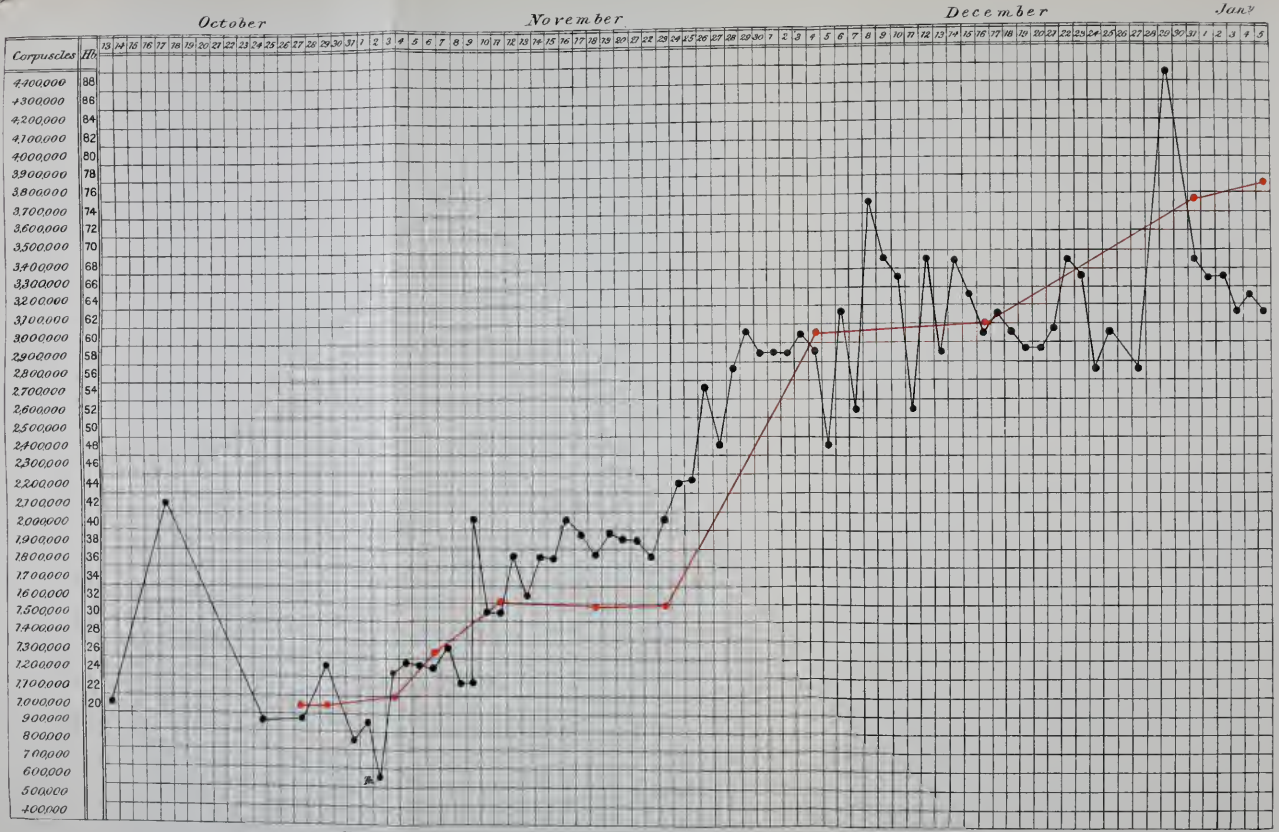
The heart was dilated, and there were all the usual hæmic bruits met with in the heart and veins in cases of extreme anæmia. Patient was much troubled with faintness, palpitation, and breathlessness, especially on exertion. The pulse was poorly filled, very compressible, and varied in rapidity from 90 to 128 per minute. The temperature was always above normal, irregular, and ranged from 99° to 101° as a rule. Beyond these facts there was nothing noteworthy in any organ.

There never had been any hæmorrhage or weakening discharge of any kind. No one who saw this case doubted that it was a typical example of pernicious anæmia. In the treatment of this patient nothing was done at first beyond careful dieting with light and peptonised foods. Even this, with the airy ward, quiet, good nursing, and rest, had a beneficial effect; for, in six days after admission, the corpuscles had risen from 1,000,000 per cmm. to 1,900,000, *i.e.*, they had nearly doubled in number. During the next seven days, however, they had fallen to 960,000 per cmm.

On *October 27th* arsenic was commenced in ℥iij. doses of Fowler's solution, increased to ℥vij. There was some response to this drug at first, but only for two days. During that period the corpuscles rose from 960,000 to 1,100,000 per cmm. Then they gradually, under the continued use of arsenic, fell to a lower figure than ever—namely, 640,000. I had determined that so soon as, under the use of arsenic, the corpuscles showed a marked tendency to fall, transfusion should be resorted to. A healthy student, Mr Scott, one of my clinical clerks, had kindly offered to hold himself in readiness.

On *November 2nd*, with the blood corpuscles at 640,000, I decided to wait no longer. Mr Duncan was sent for, and transfused into a vein in Mrs M.'s right arm 5½ oz. of blood taken from Mr Scott's arm. This blood was above the average richness in corpuscles, containing, as it did, 5,200,000 per cmm. The blood

Case II. Mrs. A. M.



The black line indicates the blood corpuscles; the red line the haemoglobin.

on this occasion was mixed with the *usual proportion of a solution of phosphate of soda*, but by some mistake the strength of the latter was made *1 in 30* instead of *1 in 20*. I may state here that this mistake can be avoided in future by taking the sp. gr., which in a 5 per cent. solution of pure phosphate of soda in distilled water is 1·031. Consequently a considerable amount of the blood coagulated in the glass vessel, and could not be used. In this way only 5⅓ oz. instead of 6 oz. of blood were injected. Owing to this coagulation, and some difficulties encountered during the operation, to which I shall again refer, it did not pass off quite so smoothly or satisfactorily as the previous ones in Mrs G.'s case. The patient had a serious faint during its performance, but was promptly brought round by the employment of suitable measures. Two hours after the operation she was looking and feeling better than before it. Her pulse was decidedly fuller, and her colour had improved. In the evening she felt much stronger. The gums, lips, and conjunctivæ were of a much redder colour, and there was a faint rosy tinge in the cheeks. The corpuscles, which before the transfusion were 640,000, now numbered 1,030,000, being an increase of 390,000 per cmm. After this there was a tolerably steady rise in the number of the corpuscles; and on November 9th, the seventh day after transfusion, the corpuscles numbered 2,080,000, *i.e., they had rather more than trebled in one week.*

The further progress in this case can be followed, to save time, on the Chart. There you can see the remarkable upward line that followed the transfusion. The corpuscles before she left the Hospital had risen to over 4,000,000 per cmm., and the hæmoglobin to 75 per cent. *Soon after the transfusion the temperature, which had ranged between* 99° *and* 101°, *fell to the normal, and there it has remained ever since.* This remarkable rise in the number of the corpuscles and fall in the temperature has been accompanied by a corresponding improvement generally. The pulse has steadily improved in volume, tension, and rate. The cheeks, gums, conjunctivæ, and finger nails have gradually assumed a rosier tint. She has also gained in strength. Her cardiac and venous murmurs gradually disappeared, and she gained about 13 lbs. in weight. About 1 lb. of this, perhaps more, must be regarded as *additional blood.* This 1 lb. of blood, with its many millions of corpuscles, must of course be added to that mere increase of corpuscles per cmm. which appears on the diagram. Not only had the number of corpuscles per cmm. and the bulk of the blood increased, but it should be noted here that *the corpuscles had,* as in the last case, *gradually assumed more normal appearances;* indeed, they became very soon *quite normal.*

Mrs M. left the Hospital on January 8th, 1889—three and a half years ago—and has remained quite well since that date.

These two cases illustrate very well the effects of repeated trans-

fusions when necessary, and of a single transfusion when that sufficed.

The next three cases I will treat more shortly.

Case III.—Mrs F., æt. 27, married; was the mother of three children. Her family and personal health history were unexceptionally good. Her circumstances altogether were comfortable, and until her present illness she had been perfectly healthy. There was no history or suggestion of syphilitic disease. Early in 1890, while she was nursing her third baby, her eldest child took ill with whooping-cough, and after an illness of seven weeks, it died. Of course this caused her much anxiety. Shortly before its death her appetite began to fail, and feelings of nausea after food developed. Sometime afterwards she began to vomit, and suffered from diarrhœa. She had no epistaxis, hæmatemesis, or hæmorrhage of any kind, and never, to her knowledge, was feverish. Gradually she got paler and weaker. Slight dropsy about the ankles and under the eyes was sometimes observed at night; and on November 5, 1890, she was sent into the Infirmary by Dr Thyne with a diagnosis of pernicious anæmia. She had been treated with iron and arsenic without avail.

On admission she presented all the characteristic features of severe anæmia, and was extremely pale, with the characteristic lemon-yellow tint of pernicious anæmia. No disease of any organ could be discovered to account for the anæmia. The blood was pale and watery looking. It flowed with great difficulty. Microscopic examination elicited the following alterations :—

1*st*, The red corpuscles were only 715,000 per cmm., a week later only 480,000.

2*nd*, There were a number of tailed and irregularly formed corpuscles.

3*rd*, Numerous microcytes—Eichorst's corpuscles.

4*th*, A number of nucleated red corpuscles, some of large size.

5*th*, The white corpuscles appeared to be normal.

6*th*, The hæmoglobin was 26 per cent.

Examination of the eyes showed remains of small hæmorrhages into the retina on the right side, and there were numerous small hæmorrhages into the retina in the left eye.

Although extremely weak, patient held her ground fairly well, and was bright and cheerful, with slight fluctuations in the numbers of the corpuscles, until November 13th, when she wandered a little; nausea came on, and she became drowsy and listless. The pulse became feeble, 123 per minute, small, very empty between the beats, and compressible.

On *November* 14 the corpuscles had fallen to 480,000 per cmm., and she was almost unconscious. It was clear that she was going to die. In this case I had—with the view of giving arsenic, diet, and care a fair trial—*delayed too long*. As, however, she had ex-

pressed herself willing to have transfusion performed if and when I thought it necessary, and having obtained her husband's permission, I got Mr Duncan to perform the operation. Only 2 oz. of blood, from the arm of Mr Macintosh, one of my students, could be injected.

Nov. 14.—Before the transfusion the corpuscles numbered 480,000 per cmm.

Nov. 15.—After the transfusion, 580,000 per cmm.

There was thus, a day after transfusion, a gain of 100,000 corpuscles, just about the same proportion as in the previous transfusions. It was, however, too late, and she gradually sank, and died on the night of the 15th. A post-mortem examination showed the characteristic appearances of pernicious anæmia, notably the large excess of iron pigment in the liver, the reddened colour of the bone marrow, together with intense pallor of all the organs, and lemon-yellow colour of the fat.

Case IV.—R. S., æt. 39, parcel postman, married, was admitted into Ward XXXII. on 10th September 1891, and is still under observation. His mother died of phthisis, a sister is anæmic, and a brother has weak lungs. Otherwise the family history is good. He drank to a considerable extent at one time, but had been a total abstainer for a year before admission. His present illness would appear to have been mainly caused by great irregularity in the hours of breakfast and work. *It began, as in all the other cases, with symptoms of dyspepsia.* Gradually, during the last two years, he has been getting paler and weaker, and when he came into the Hospital he weighed only 8 st. 13½ lbs. He was extremely bloodless, pale, and flabby looking—indeed, he presented the aspect of extreme anæmia, with the lemon-yellow tinge, which is so characteristic of pernicious anæmia.

On *September 25th* the corpuscles numbered 1,200,000 per cmm. They fluctuated up and down about this point in spite of the use of such drugs as iron and arsenic, and carefully regulated diet.

On *December 16th* they had fallen to 920,000 per cmm., and as he was extremely weak, and warned by the last case, I feared that so prolonged an extreme anæmia might lead to incurable degeneration of the tissues generally, I determined to employ the transfusion of human blood without further delay.

On *December 17th* Dr Duncan transfused from the arm of Mr Webster, one of my clinical clerks, 5½ oz. of healthy blood. Seven hours after transfusion the blood was examined, and the corpuscles, which before transfusion numbered 920,000, were found to have risen to 1,110,000 per cmm., being a rise of 290,000 per cmm. The further results of the transfusion may be summarized as follows, and showed a steady rise in the corpuscles for more than three months:—

The average for the 12 days after this first transfusion was	1,173,000
For January 1892,	1,240,000
For February,	1,440,000

On *February 15th* they were 1,570,000, *i.e.*, 650,000 more than they had been before transfusion. But this does not represent all the gain. During this period he had *gained greatly in strength, and had put on 9 lbs. additional weight*, which means about 10 oz. of additional blood teeming with millions of millions of red corpuscles beyond what is shown by their mere increase per cmm.

For the next three months he remained very much in *statu quo;* indeed, although the increase in his weight was maintained, the corpuscles showed a tendency to diminish again. We therefore determined to repeat the transfusion. On *March 9th* he himself was eager to have the operation repeated, so grateful was he for the result of his first transfusion. The corpuscles at this time were 1,280,000 per cmm. $4\frac{3}{4}$ oz. of blood from the arm of Mr Sandeman was mixed with $3\frac{1}{4}$ oz. of the phosphate of soda solution, and of this about $7\frac{1}{2}$ oz. were injected, *i.e.*, a little over 4 oz. of blood. Now, although the operation in this case was most satisfactorily performed, a double error was made in the preliminary state of preparing the solution.

1*st*, The distilled water—obtained as such from the chemist's department in the Infirmary—with which the solution was prepared was found afterwards to have been *distilled some months before.* It was quite opalescent, and contained a quantity of flocculent-like substances floating through it.

2*nd*, The solution had not been, as usual, *twice brought to the boiling point before being used.* These mistakes were, of course, not found out until afterwards, but they fully accounted for the unsatisfactory immediate results in this case, which were as follows:—

About fifteen minutes after the transfusion was over, *i.e.*, at 1·30 P.M., patient had a severe rigor, with vomiting and intense pain in the stomach; his pulse, which had fallen during the operation from 84 to 72, became irregular. At 2·15 P.M. his temperature had risen from normal to 104°. His urine became bloody-looking, and was found by Dr Noël Paton to contain a considerable amount of hæmoglobin, but no blood corpuscles. During the next day or two the right arm, where the injection had been made, became sore, and he complained of pain in the axilla. A *herpetic* eruption appeared on right upper eyelid, which was also swollen and red, and on the lower lip. This eruption soon became *pustular.* There could be no doubt that poison of some kind had been injected. The arm afterwards became intensely œdematous, and there was pain along the line of the veins above the seat of the transfusion. I need hardly say that the immediate effects of the transfusion were greatly interfered with by this feverish attack. The result on the

corpuscles was that, whereas before the transfusion the red corpuscles numbered 1,280,000 per cmm., and the hæmoglobin 27 per cent., seven hours after the transfusion the red corpuscles had fallen to 1,190,000 and the hæmoglobin to 25 per cent.,—there was thus a fall in the corpuscles to the extent of 90,000 per cmm.

The temperature fell considerably on the evening of the 9th, the day of transfusion, but remained irregular for several days, reaching 102° on the 12th. Then it and the other feverish symptoms gradually subsided, and simultaneously the corpuscles began to rise again. They, indeed, gradually rose to about the same average as that preceding the transfusion, and fluctuated about that point till about the end of April, when they again began to rise higher. The average of seven observations taken between the 1st and 24th of May gives 1,770,000 corpuscles per cmm. His general health has greatly improved, and he is still gaining in weight.

When he came in he weighed . .	8 st. 13½ lbs.
Now he weighs	9 „ 9 lbs.

It is my intention to have him transfused again very soon, and I have no doubt that by rigid attention to every precaution we will have a thoroughly satisfactory result in this case.

Case V.—T. B., æt. 63, church officer, married, came of a healthy stock, and was himself quite well until October of last year (1891). His present illness began in this way. One day when he was very busy he missed his lunch; indigestion and nausea came on, and from that time he gradually became weaker and paler, till he was sent into the Infirmary under my care by Dr William Russell on March 5th, 1892.

Iron and arsenic, and arsenic alone, had been administered before his admission without effect, and as he had rapidly been going down hill latterly, was very weak, and his corpuscles on admission numbered only 840,000 per cmm., it was determined to transfuse without delay.

On *March 9th* the corpuscles numbered 890,000 per cmm., and on that day he was transfused. Mr M'Nicoll, one of my students, gave his blood, and the solution employed was the same faulty solution which was used in the second operation in R. S.'s case,—indeed, both operations were performed on the same day, T. B.'s being taken first. Owing to coagulation of the blood in the vessel only 1⅓ oz. of blood were injected. There was thus a smaller amount of both the solution and of the blood than in any other operation. Patient stood the transfusion well; his pulse fell during the operation from 100 to 92, and remained steady and full. Temperature at 12, before the operation, was 98°, at 1 P.M. it was still normal. A quarter of an hour after the operation he had a severe rigor which lasted 50 minutes. Temperature at 2 P.M. was 102°. The pulse was 124, respirations 18 per minute.

By 9 P.M. the temperature had fallen to 98·2°, and there it remained. There was no swelling of the arm or secondary eruption or complication in this case, such as we had in S.'s case.

The effects of the operation in this case were as follows:—

Before transfusion the blood corpuscles were	890,000	per cmm,	Hb. 11%
After transfusion the blood corpuscles were	930,000	„	Hb. 13%
Increase	40,000	„	2%

From this time onwards there was a very steady, rapid increase in both blood corpuscles and hæmoglobin. And by April 30th, *i.e.*, 52 days after the transfusion, the corpuscles numbered 4,200,000 per cmm., and the Hb. 54 per cent. His weight had also risen from 8 st. 10½ lbs., which it was on the day of transfusion, to 9 st. 6 lbs., *i.e.* an increase of 9½ lbs. He was altogether better and stronger; this was acknowledged by him and recognised by all who saw him.

It is difficult to account for the termination of this case.

From the time of the operation until the 2nd April, *i.e.*, for nearly four weeks, the temperature remained normal. On April 2nd it rose on one day from 97°·8 to 102°·8, and thereafter remained irregularly pyrexial.

On *April 23rd* signs of consolidation with crepitations were noted in the left sub-clavicular region. Up to this date the corpuscles and hæmoglobin had steadily increased, and his weight had been maintained. From this date onwards the course of the case was that of a rapidly progressive phthisis, and he died on May 16th. To the end, however, the corpuscles remained over 4,000,000. The post-mortem showed marked diffuse tuberculosis of both lungs.

That the anæmia was not tubercular may be taken for granted; for in tubercular phthisis the proportion of *red corpuscles per cmm. is as a rule—indeed almost invariably—normal or above the normal*, while the *total bulk* of the blood and of the corpuscles is *greatly reduced*. In this case *the anæmia was rapidly disappearing, and the bulk of the blood increasing*, when the symptoms and signs of phthisis appeared.

Was this man infected in the wards, either from the injected blood or through the atmosphere? We cannot positively answer this question. The sad termination does not negative the fact that he had during the interval following the transfusion almost recovered from his pernicious anæmia.

I must ask your indulgence for a little while I gather together what is to be learned from these cases, and I will do so under the following heads:—

First, The results anticipated from transfusion of human blood, and those actually obtained when the operation was in all respects satisfactorily performed.

Before operating at all the question we put to ourselves was this, What is the utmost that we can expect from the injection into the veins of a patient suffering from pernicious anæmia of a definite amount of human blood? My belief was, and still is, that, with proper precautions, blood equal in vitality to living blood might be injected into a human being's veins. I further believed that the admixture of a considerable quantity of living healthy blood with the blood of the diseased individual might powerfully influence the diseased blood with which it became mixed, increasing its vitality and resistance to destructive influences, and that it might also beneficially affect the blood-forming organs, tending to a restoration of their blood-forming power. For the purpose of testing this point we attempted, from the weight of the patient's body and the relative amount of the blood—taken as about 1-13th of the body-weight—to estimate approximately the proportionate number of blood corpuscles that would be added to each cmm. by the injection of a definite amount of normal human blood. This probable addition we determined to compare with the actual results obtained at a considerable interval after transfusion. Thus we calculated in the case of Mrs G., who weighed 6 st. 3 lbs., that by the addition of 6 oz. of normal blood we should gain an addition of about, indeed a little over, 300,000 corpuscles per cmm. Now, although we had made this calculation, we were exceedingly surprised to find that, from the first transfusion, we had obtained an addition of 310,000 corpuscles per cmm. That this was not a mere accidental and pretty coincidence has been shown by the fact that nearly *every properly performed subsequent transfusion not complicated with fever has been followed by a like proportionate increase in the number of the corpuscles.* The inference which one cannot avoid drawing is that all the red corpuscles in the blood of a healthy donor are really added to the blood of the recipient without any perceptible destruction of them in the process. Let me recall the immediate results of the different transfusions so as to illustrate this point.

1*st Transfusion.*—In Mrs G.'s case,

Before operation,	1,160,000	per cmm.
After „	1,470,000	„
Increase,	310,000	„

2*nd Transfusion.*—Now I must remind you that Mr Hardyman's blood, which was used in this operation, was very rich in corpuscles, there being 5,500,000 per cmm. We therefore looked for a greater addition of corpuscles than in the first operation. These are the facts.

Before operation,	1,300,000	per cmm.
After „	1,670,000	„
Increase,	370,000	„

3rd Transfusion.—This was performed when the corpuscles *showed a decided tendency to progressive increase.* The result is very interesting, for we got much more than could be accounted for by the mere addition of the blood.

Before operation,	2,215,000	per cmm.
After „	2,820,000	„
Increase,	605,000	„

Part of the rapid increase of the blood corpuscles must be accounted for by an impetus given to the blood-forming organs on the one hand, and to the already improving blood corpuscles on the other, increasing the resistance of the latter.

4th Transfusion.—In Mrs M.'s case, weight almost the same as Mrs G.'s, 6 st. 7 lbs.

Before transfusion,	640,000	per cmm.
After „	1,030,000	„
Increase,	390,000	„

In this case the *donor's blood was extremely rich in red corpuscles.* Thus we see that in these cases of pernicious anæmia, the body weight being 6 st. 3 lbs. in the one, and 6 st. 7 lbs. in the other, the injection into the vein of 6 oz. of healthy blood was calculated to add, and actually did add, about 300,000 red corpuscles per cmm.

5th Transfusion.—Mrs. F., weight 5 st. 5 lbs. Rather less than 2 oz. of blood, instead of 6 oz., were added in this case.

Before transfusion,	480,000	per cmm.
After „	580,000	„
Increase,	100,000	„

This was just what might have been anticipated from the injection of rather less than a third of the amount of the blood injected in the previous cases.

6th Transfusion.—R. S., weighed 8 st. 13 lbs., and therefore from a similar amount of blood a smaller rise was to be expected.

Before transfusion the R. B. Cs. were	920,000	per cmm.
After „ „	1,110,000	„
Increase,	290,000	

7th Transfusion.—T. B., weighed 8 st. 10 lbs.; owing to coagulation of blood in the dish received only about $1\frac{1}{2}$ ozs.

Before transfusion,	890,000	per cmm.
After „	930,000	„
Increase,	40,000	„

This operation was, however, unsatisfactory, and will be referred to again. But the fact that all the blood corpuscles were added to,

and remained circulating in the blood for a period of not less than two or three days—important though it undoubtedly is—was by no means the most satisfactory result gained.

Most of these transfusions *were followed by a progressive rise in the number of the blood corpuscles above the number added by transfusion.* This is the most satisfactory point of all. This result was noted after all the four transfusions in Mrs G.'s case,—most strikingly after the second and third operations.

In *Mrs M.'s* case the ascent was rapid and striking up to complete recovery, after only one transfusion. In *R. S.'s* case there was a steady rise for several weeks after the first transfusion, and a postponed rise after the second unsatisfactory operation. In T. B's. case, although a serious mistake occurred, to which we shall have to refer immediately, his corpuscles rose rapidly after his first transfusion, reaching 4,200,000 on April 30th, *i.e.*, fifty days after transfusion.

Second, The lessons to be learned from our mistakes.

1*st*, Unless great care is taken in the preparation of the solution and the instruments and vessels employed, injury may be done to the patient. I have no doubt the rigors, fever, phlebitis, herpetic eruption, and destruction of corpuscles in connexion with the second operation in R. S.'s case was the result of the impure distilled water used, and the failure to boil the solution previous to its employment. *We had no fever in any of the satisfactory transfusions.*

2*nd*, The substitution of a too strong solution of the phosphate of soda may cause a fall instead of a rise in the number of the corpuscles. This was illustrated in the fourth transfusion in Mrs G.'s case, in which a ten per cent. solution was employed instead of the five per cent. solution. The result was a slight fall, instead of the usual rise in the number of the corpuscles.

3*rd*, A too weak solution, or an insufficient amount of the solution, may allow coagulation to occur, and either cause the operation to be suspended before all the blood is injected, or endanger the patient's life. No accident has ever happened in my cases from this cause, but in one or two instances much valuable blood has thereby been lost.

4*th*, A too rapid injection of the blood may cause dangerous, and possibly even fatal consequences. This was exemplified in my most successful case—Mrs M.—in which the end of a syringeful was injected to save the blood after the pulse began to show irregularity of beat. This was followed by alarming syncope and other symptoms, which under prompt treatment rapidly disappeared. At this point I should remind you that in this disease the whole vascular system has accommodated itself to a greatly diminished volume of blood. Hence the sudden introduction of 6 or 8 oz. of blood into the contracted vascular system causes a marked alteration of pressure in the heart and lungs not without risk. The condition here is very different from that

present in anæmia from rapid loss of blood,—as, *e.g.*, in surgical cases where the vessels are large and empty. In the latter cases rapid transfusion can be borne with safety because it improves the blood-pressure.

5*th*, For the purpose of rendering the operation absolutely safe, I think the operator should have a table of rules placed in the hands of his assistants, and should in each case have before operating the definite assurance that all of them have been carefully attended to.

6*th*, One of the most important lessons which I have personally deduced from my experiences is this:—In none of my cases, even where a distinct mistake has been made, has any accident leaving any permanent effect happened: nor has any other than a good result ultimately followed the transfusion in any of my nine transfusions. Hence I must conclude that, *given the requisite attention to all details, and the necessary surgical skill, the operation cannot be regarded as a dangerous one*—it can therefore be recommended with confidence in so perilous a disease when all other remedies have failed.

In making this statement I do not forget how much the success of these transfusions has depended on the consummate skill and patience of my esteemed colleague Dr John Duncan. It is a delicate operation, and that it should not be attempted rashly by any one but an expert is clear; but I doubt not we will always be able to find experts, in Edinburgh and other large cities at least, when the operation is judged necessary.

The conclusions at which I have arrived from my experience of transfusion of human blood in pernicious anæmia may be very briefly summed up as follows:—

1*st*, If all the necessary precautions are strictly adhered to the operation is perfectly safe.

2*nd*, Quite healthy blood with living blood corpuscles can be added to the diseased blood of the patient.

3*rd*, This blood exerts a beneficial influence both on the blood with which it is mixed and on the blood-forming organs, for—

(*a.*) Sooner or later the abnormal peculiarities in the forms and the exceptional varieties of the corpuscles disappear.

In Mrs G.'s and Mrs M.'s cases the corpuscles became normal in those respects very shortly after the first transfusion.

(*b.*) Sooner or later the blood corpuscles begin to increase in excess of those added by the transfusions.

4*th*, These facts are opposed to the view that an abnormal destructive activity of a disordered liver is the only or the main pathological condition in pernicious anæmia; for it is difficult, were this true, to understand how the blood corpuscles added should not soon fall victims to the destructive influence of the liver cells.

The foregoing results of transfusion rather favour, although

they do not absolutely shut one up to, the conclusion that the true pathology is, as I have already said, probably somewhat like this:—

1*st*, The real condition of the blood in pernicious anæmia is a delicacy and tendency to early death of the red blood corpuscles.

2*nd*, The probable starting-point of this delicacy and feeble resistance in the blood corpuscles is some functional weakness in the blood-forming organs, which may be due to various possible causes.

3*rd*, The irregular-shaped, variously-sized, and otherwise abnormal blood corpuscles point to some such imperfect genesis.

4*th*, Consequently without any abnormally increased destructive force in the portal system and organs—it being a normal function of the liver cells to destroy the red corpuscles—a greatly increased death-rate of these delicate and short-lived corpuscles takes place.

5*th*, The introduction by transfusion of a considerable amount of healthy blood acts beneficially in a twofold way:—

(*a.*) By immediately improving the health and resistance of the blood (including the delicate blood corpuscles) which becomes mixed with it; and

(*b.*) Later on, by gradually operating beneficially on the blood-forming organs through which it circulates, restoring their blood-forming functions to the normal condition.

Mr President and Gentlemen, I must apologise for the great length of this paper. Long as it is, I have been obliged to omit many interesting facts contained in my reports and details, and which I propose to give at a future date in a supplementary paper.

Dr Affleck said that having been invited to take part in the discussion, he thought he could perhaps best do so by giving the Society a brief account of a case of pernicious anæmia recently under his care in the Royal Infirmary, in which transfusion was followed by the most marked success, and by exhibiting the patient. W. L., æt. 44, a carter, was admitted to Ward 29 on 4th January last with all the symptoms and signs of anæmia in an aggravated form. He had been under treatment in the Royal Infirmary in May last for similar symptoms, and at that time there was a suspicion of Addison's disease, but after a long stay in the hospital he left somewhat improved, and had continued fairly well until some weeks prior to his admission on the present occasion. No cause could be discovered for the profound anæmia which now existed. On examination of his blood the red corpuscles were found to be reduced to 1,240,000 per cmm., and they were of extremely irregular shape, showing also numerous megalocytes and microcytes. The hæmoglobin, which was 20 per cent., was fairly well represented in each corpuscle. There was no absolute

increase in the number of leucocytes. The patient complained of pain in the chest, cough, shortness of breath, and great exhaustion. The skin was extremely pallid, and had the characteristic lemon tint. Cardiac, pulmonary, subclavian, and jugular murmurs were well heard. There were no retinal hæmorrhages. The patient was put upon treatment by arsenic, and his diet was adjusted to his feeble digestive capacity. Day by day all through his stay in the hospital the blood corpuscles were carefully counted, and the calculation was duly checked. During the whole of January the condition remained without much change, although the tendency was downward, the corpuscles falling frequently to 1,000,000 and even lower. As February advanced a change for the worse became very apparent. The corpuscles continued to diminish, being sometimes as low as 970,000, and the hæmoglobin 17 per cent. It would be difficult to convey any adequate idea of the state of utter and seemingly hopeless exhaustion to which this patient was reduced by the middle of February, and it seemed unlikely that he could survive for another fortnight. He (Dr Affleck) had seen many fatal cases of pernicious anæmia, and this case had every character of one of that class. At this time transfusion was proposed to the patient, with considerable hesitation on his (Dr Affleck's) part, as a last resort, and the patient, who felt himself going down rapidly, gave his consent. Mr John Duncan was communicated with, and he agreed to perform the operation, which was accordingly done on 17th February. Mr Duncan would doubtless refer to some of the details of the process in this particular case, but he (Dr Affleck) as a witness of the proceeding could not avoid paying a tribute to the great care and patience with which the operation was performed in circumstances of special difficulty. The blood was generously furnished by one of the clinical clerks, Mr A. Scot Skirving, and it was treated in the usual way with a solution of sodium phosphate. The patient was extremely weak, and he was very timid, and bore the operation rather badly, frequently complaining of pain in the back, and asking to get done with it. After about four ounces had been injected, the operation, which occupied over an hour, from the slowness with which it was necessary to proceed, had to be stopped, the patient becoming restless. His pulse, however, had kept good and even improved during the operation, and there were no alarming symptoms present. About two hours after the transfusion the patient had a rigor, and the temperature rose to 103°. This feverish symptom passed off in the course of the night, and the following day he was feeling pretty well. No increase, however, was discernible in the corpuscles, which, on the contrary, fell to 790,000, the lowest point yet observed. Four days after the operation (21st Feb.) a change for the better was quite manifest in the patient. He felt stronger and better, and had a good appetite, while the blood corpuscles rose to 1,010,000. This proved to be the beginning

of a steadily progressive increase.[1] When the patient left for the convalescent house on 14th April his red blood corpuscles numbered 4,900,000, and on his coming in to the Infirmary to report himself on 21st April, they had risen to 5,110,000, and he had a ruddy, brown appearance of strong health. The patient returned home perfectly well, and has continued so till the present time. He (Dr Affleck) sent for him yesterday, June 22nd, and found, notwithstanding the fact that the poor man has not succeeded in falling in with work, and therefore has not been so well fed as he might have been, the red corpuscles still number about 5,000,000 and are of perfectly normal appearance, and the members of the Society would have an opportunity of judging by the patient's appearance of the completeness of the recovery in this case. (Here Dr Affleck exhibited the patient.) No doubt in a case of this kind a relapse might occur, but there is no appearance of any such thing, and it is, to say the least, a highly satisfactory result to have an improvement so striking as this. Specially noteworthy are the smallness of the amount of blood used, and the rapidity, steadiness, and completeness of the progress back to the normal character of the blood. Whatever its *modus operandi*, this small transfusion must have had the effect of arresting blood destruction and restoring healthy hæmopoiesis. To regard the improvement in this case as a mere coincidence would be to shut one's eyes to the facts of its clinical history. A result like this, and like those narrated in the interesting communication of Dr Brakenridge, would seem to show that there are cases—it may be classes of cases—of this disease where transfusion is the one sovereign remedy, and at all events they go a long way to justify the continuance of this proceeding as a rational means of treatment. The case now narrated has impressed upon him (Dr Affleck) a lesson in this direction. He confessed to having had a poor opinion of the operation from all he had observed and read of it, but he has entirely changed his view, and while, of course, in any case he should in the first instance resort to the usual measures of treatment by arsenic, etc. (under which he has seen several cases greatly benefited), and give these every opportunity, he should still feel that he had a further resource which might stand between the patient's malady and death, and should urge the employment of it with some measure of hope.

[1] The following figures will show the rate of progress :—

Feb.	24.	1,410,000	March	13.	3,880,000
„	26.	1,920,000	„	20.	4,180,000
„	28.	2,150,000	„	29.	4,190,000
March	1.	2,440,000	April	14.	4,910,000
„	4.	3,160,000	„	21.	5,110,000
„	10.	3,370,000			

A daily observation was made. Some days showed a slight falling off, but the above figures illustrate the progressive character of the improvement following the transfusion.

Mr John Duncan said he would not venture to discuss the pathology of pernicious anæmia, or whether the remarkable results which had been detailed by Dr Brakenridge and Dr Affleck were due to one or other constituent of the blood, or in consequence of an action on pathological or physiological functions of the body. He (Mr Duncan) concerned himself solely with the surgical aspects of the treatment which was adopted. It is within the experience of all surgeons to be called upon to consider the propriety of introducing fluid into the circulation in cases of impending death from hæmorrhage. In most cases, the hæmorrhage being arrested either artificially or by the syncope of the patient, recovery even after extreme losses was comparatively easy and speedy; so that there were really few of which it could not be said that if the hæmorrhage were arrested the patient would recover; if it were not arrested, treatment would not avail. Still those few remain, and there were still others in which the danger of shock from the injury, accidental or operative, was seriously aggravated by loss of blood. In such cases he had from time to time practised the various operations that have been advocated, such as the injection of saline solution, of milk, of defibrinated blood, and of human blood in its entirety. He had been greatly dissatisfied with all. He had found the direct injection of blood difficult and dangerous, and had been impressed by the fact that while a saline solution frequently produced a remarkable rally, the patient invariably died within four-and-twenty hours. In view of a recent paper, which gave the satisfactory effect of injecting larger quantities than he had done of normal saline solution, this opinion may after trial require to be modified. Such, however, had been his unfortunate experience when, some years ago, his attention was directed by Mr Cotterill to the suggestion made by Dr Pavy to maintain the blood fluid by means of phosphate of soda. He lost no time in putting this in practice, and the results obtained were so encouraging that he extended the application of the method to the reintroduction of the blood lost into a vein on the surface of the wound in cases of surgical operation where the hæmostatic methods employed might be tardy and difficult. It was not necessary to recapitulate instances, which have been numerous, of life directly saved by this method. He would only refer to the case in which Dr Edward Carmichael saved a patient by injection of blood from his own arm, and to the large quantity of twenty-two ounces of blood and soda solution which he (Mr Duncan) reinjected with the best results in a patient of his colleague Mr Miller. In a former paper in the *Edinburgh Medical Journal* he had explained the method and the necessary precautions. They were so simple that they did not need to be repeated, and he would only direct attention to certain points in connexion with this farther extension of transfusion of blood to cases of anæmia at the suggestion of Dr Brakenridge, Dr Affleck, and others. He desired, however, first to insist on what he emphasized in his

former paper,—the necessity for the utmost attention to asepsis. He then recorded a case of septicæmia, in which there was reason to fear that contamination of the fluid by septic pus had been allowed during the collection of the blood as it flowed in a case of amputation high in the thigh. He took blame to himself that a certain measure of septic infection occurred in the two cases done at the same time, to which Dr Brakenridge had referred. They were in the habit of keeping in the wards, in a large flask stopped with cotton wool, a carefully asepticised solution of phosphate of soda in distilled water. Unfortunately this had been recently used up, and, when he ordered the apparatus to be prepared, a fresh solution was simply ordered from the laboratory without the necessary directions for repeated boiling which he had given in his paper. In a matter of this kind he ought to have personally attended to every detail, but he was under the impression that the person to whom it was entrusted had been cognisant of the method followed in former operations. These precautions were necessary in all transfusions. But there was one peculiarity in cases of anæmia for which experience in infusion for hæmorrhage did not prepare one. He meant the necessity for extreme slowness in operating. No doubt it was always advisable not to be too rapid, but in pernicious anæmia the sensitiveness of the heart and vessels to increased tension is doubtless increased, while there cannot, of course, be the diminution of pressure, the room, which obtains after hæmorrhage. It was the fact, at least, that, with variations in individuals, irregularity of pulse, breathlessness, and pain in the small of the back, were easily produced by injecting with rapidity, and those operations were most satisfactory which were done most slowly. It was an evident corollary from this that the amount of the solution should be sufficient to keep the blood fluid for a considerable time. He regarded 30 minutes as the minimum time that should be occupied in injecting 8 ounces of fluid, and while the coagulating tendency of the blood varied considerably, he should say that one part of solution to three parts of blood was rather small to insure perfect fluidity, that the best proportion was one-third, and that a much larger quantity of the solution might be used without diminishing the benefit. It was common after injection, but not by any means invariable, to have a rigor within a few hours. He had convinced himself that this was not connected with any septic state. Its exact meaning had still to be made out, but it might be associated with the unavoidable mechanical damage which must be done to some of the corpuscles in the progress of the operation. This was also indicated by the hæmoglobinuria which was sometimes present, and Dr Gillespie had pointed out to him (Dr Duncan) the extreme tendency to rigor in paroxysmal cases of that condition. It had been maintained that the injection of blood was useless, and that the blood-cells thrown in were no more viable when human than when animal blood had been used. The rigors and hæmaturia had been

adduced in support of this opinion. But the cases related by Dr Brakenridge seemed conclusively to negative this hypothesis. Moreover, the hæmoglobinuria never corresponded to the quantity of blood injected, and both it and rigor were often entirely absent. Whatever constituent of the blood might be useful in these cases of anæmia, it was permissible to believe—(1), that most of them when injected were still viable; (2), that, as with skin, bone, and nerve, healthy blood tissue might have a determining influence in producing its like; (3), that the introduction of blood in cases of hæmorrhage was more valuable bulk for bulk than that of saline solution; and (4), that these cases proved that with little risk a beneficial effect in anæmia was produced by the operation.

The President, after remarking on the extreme interest and importance of the communications just made, ventured to hope that the discussion which would now follow would be also instructive.

Dr James said he wished, in the first place, to express his sense of the great interest and value of Dr Brakenridge's paper and of Dr Affleck's addition to it. Without in any way diverting attention from the important points connected with the practical application of the treatment by transfusion in this disease, he (Dr James) thought they were now in a better position than they had ever been before to consider why transfusion of blood should be so frequently found to be of the immense value which it showed itself to be in the case they had heard of to-night. How, for example, could the transfusion of such a small quantity of blood—in Dr Affleck's case only 4 oz.—arrest completely the tremendous loss of corpuscles which had been occurring? Now he (Dr James) thought they could not suppose that the corpuscles could have had, as corpuscles, much to do with it. What he thought they might suppose was, that in pernicious anæmia there was something in the system, if he might use the term, *micro-organismal* in character, whether it was derived from the alimentary canal, whether it produced the anæmia by giving origin to unhealthy young red corpuscles, or by killing off too soon healthy old ones, or by both, it did not for their present purpose signify. Given this morbid substance, it produced, month after month and week after week, more and more anæmia. Now, they could explain the value of a single small transfusion, if they supposed that healthy blood could exercise a phagocytic action on this morbid substance. For this they had the analogy of Koch's tuberculosis, of the use of thyroid juice injections in myxœdema, and still more aptly of the treatment of tetanus by the injection of the serum of an animal which had been rendered immune to that disease. What he (Dr James) would wish to hear discussed, therefore, by the Society was the probability of the good effects of transfusions being explained in this way. Further, if this was the explanation, they could understand that a rise of temperature after the operation might be of the same nature as the reactions after a tuberculin injection. In Dr Affleck's case

this rise was well marked, and the patient seemed none the worse, perhaps even all the better, of its occurrence. Lastly, if there was anything in this explanation, the injection of healthy blood serum should be as efficient as the transfusion of healthy blood.

Dr William Russell hoped the Society would permit him to refer to a paper he had read on Pernicious Anæmia to the British Medical Association in 1888, and which was published in the *British Medical Journal* in 1889. When he began to study the disease, the question which presented itself to him was whether it was the result of diminished formation or of increased destruction of red blood corpuscles, and at that time there was little guidance to be found in the literature of the subject on that point. When examining the condition of the organs he had found that there was evidence of great blood destruction in the liver, spleen, and kidneys; and similar observations had been made by others. On finding this marked evidence of blood destruction, he was attracted by the view that pernicious anæmia might be a liver disease, but he soon abandoned that view for various reasons. While there was this undoubted evidence of greatly increased destruction on the one hand, there was to be placed along with it the further fully accepted fact of a great increase in the red marrow of the bones, and his contention in the paper referred to was that this was to be regarded as a hyperplasia of a tissue which was accepted as an important blood-forming tissue. There were thus two facts established,—increased blood destruction, which he did not believe was due to what he then called a blood-hunger on the part of the liver; and an increase of blood-forming tissue, which he regarded as a compensatory hyperplasia. Further than that it seemed to him then that neither physiology nor pathology permitted them to go. The question now was whether any additional light had been thrown on this subject by Dr Brakenridge's work, and by work in other fields, and he thought there had been. Dr Brakenridge's therapeutic experiments, coupled with Dr Affleck's case, clearly and indisputably established the fact that after transfusion the blood continued to increase in corpuscles and hæmoglobin long after the corpuscles added by the transfusions must have perished. This fact was of the greatest interest and importance, in view of the work which had been done in this limited period of time by Metchnikoff and many others, working on parallel lines, all tending to place serum therapeutics in the position of a new science. There was abundant evidence now of the influence of blood serum under certain conditions to counteract the effect of bacterial poisons, no matter whether we regarded that action as an antitoxic one or as influencing the phagocytic action of certain cells. From these facts and from Dr Brakenridge's work, it appeared to him that a further provisional step might be taken in the explanation of the disease, for there was no reason why pathology should not avail itself of therapeutic facts, and the view which had been before his

mind for some time, knowing as he did the results Dr Brakenridge was obtaining, and in view of other facts with which he was acquainted but could not refer to, was in the direction Dr James had suggested, namely, that the disease was an infective one, using that word in its widest sense. This view fitted in with the results of the therapeutic experiments by transfusion, and if this surmise were correct, Dr James's further suggestion that the blood serum might be enough was also probably correct. The view that the transfused blood acted beneficially on the blood-forming tissues did not appeal to him as it did to Dr Brakenridge; whereas the view that the transfused blood counteracted a poison was in harmony with recent work. The question still remained, whether this poison was a ptomaine absorbed from the alimentary canal, as Hunter held, or a poison generated by an organism within the blood itself. The former view did not commend itself to him, nor did he think that the results of transfusion supported it, more especially those cases where a single transfusion had proved sufficient.

Dr Smart, referring to clinical work which he had done in relation to the treatment and pathology of anæmia, said that he had been particularly interested in one feature to which his attention had been specially drawn, and which, he thought, was of immediate interest in relation to the present discussion. He had obtained a striking hæmatine reaction in the urines of all the severer cases of bad anæmias by means of a reagent first suggested to him by MacMunn[1] in his book on the *Clinical Chemistry of the Urine*. There is no ocular evidence of blood in these urines, many of them being pale and colourless. The characteristic red colour only appears with the reagent. The view explanatory of this evidence of blood in the urine is that ptomaines, during the course of anæmia, are developed in the bowel from secondary fæcal decompositions, and, being absorbed into the blood, are destructive of the red corpuscles to the extent of causing anæmia. The results of examinations of a number of his cases of anæmia, including those of chlorosis and pernicious anæmia, tended to corroborate this view. He further took opportunities of examining the urines of patients in whom there had been lengthened retention of fæces on account of chronic constipation. And in one case of intestinal obstruction with fæcal retention for nearly a month, the urine exhibited the hæmatine reaction most strikingly—the patient, he may add, being at the time profoundly anæmic. If the view be correct, and these results would imply that it was well founded, that in anæmia, especially of the pernicious type, the red corpuscles in the circulation undergo destructive changes in consequence of auto-blood-intoxication in the way described above, it may be held

[1] MacMunn, in the statement referred to above, is, I find, adducing proof in support of Sir Andrew Clark's theory, that in chlorosis and allied conditions fæcal matters are absorbed from the intestine into the blood, which have a rapidly destructive effect upon the red corpuscles.

as most probable that the blood-making organs, as well as the entire range of tissue, activity suffer from the effects of this sepsis. This is evident, especially in pernicious anæmia, by the great numerical depreciation of the red corpuscles, their altered shapes, and the low standard of hæmoglobin—all indicating the disability of the blood-making organs to supply the number and quality of the corpuscles required for the needs of a healthy vitality. If we take into account this twofold disability, we may better appreciate the results which followed the transfusion of healthy blood as mentioned by Dr Brakenridge, and as seen in Dr Affleck's patient, into whom only four ounces were injected. The remarkable effects produced in these cases cannot, on any view, be explained by reference to the quantity of blood used. It would be more consonant with our knowledge and experience to ascribe these effects to the energetic action of the healthy blood acting under the special circumstance as a most potent therapeutical agent, effectually stimulating the organs and functions, depraved and devitalized to the last degree for want of it, and rousing them into renewed and sustained activity by affording the tissues, and still more the great trophic and other centres immediately concerned in blood-making and nutrition, that kind and degree of stimulation most efficacious. In illustration of the extraordinary effects of powerful reagents, he instanced those following injection of minute quantities of tuberculin upon tubercular conditions. Dr Brakenridge's work on transfusion was of great value in settling the question of its eligibility as a means of treatment. After the results obtained we must now regard it as a remedy of great value, and one which it will be our duty to give our patients the benefit of.

Dr Robert Muir considered that such cases as those brought forward by Dr Brakenridge and Dr Affleck were of great importance in relation to the pathology of the disease, as the results appeared too striking to be explained as coincidences merely. Yet cases of pernicious anæmia sometimes pursued a very remarkable course when not treated by transfusion. He mentioned a case in which during observation the number of corpuscles somewhat rapidly fell to 500,000 per cmm., and in which, owing to arsenic being badly borne by the patient, the only treatment was small doses of iron, which could scarcely have had any part in bringing about the result. The number of corpuscles, after falling to the figure mentioned, suddenly began to increase, and steadily rose till it reached nearly 4,000,000 per cmm., when the patient left the hospital. Such a case seemed more easily explained on the theory that a process of blood destruction was going on, and that the poison causing it had in some way been suddenly got rid of, the normal or hyperplastic bone marrow then being free to produce a rapid increase in the number of red corpuscles. If the pigment containing iron found in the liver in such cases was due to a rapid natural death of weak corpuscles, one would expect to find

it in severe cases of secondary or symptomatic anæmia, which, so far as his experience went, was not the case. Moreover, the corpuscles in pernicious anæmia were on the whole larger and better coloured than in cases of secondary anæmia, *e.g.*, due to cancer. He considered that the theory of blood destruction explained most of the clinical and pathological facts, though some were still left unexplained. If in some cases the blood transfused produced a marked improvement, he thought that the theory suggested by Dr James was the more probable one. In Dr Affleck's case the quantity of blood transfused on one occasion only was so small that it was scarcely conceivable that the *red corpuscles* could bring about such a change in the blood-forming apparatus as to produce the effects observed. If, on the other hand, some chemical poison, possibly a bacterial product, caused the breaking down of red corpuscles, the blood-serum of a healthy individual might possess, in some cases, an antagonistic action. Somewhat analogous facts were known with regard to certain diseases, *e.g.*, tetanus and diphtheria. If the serum did possess such a property, it might possibly be found to be more powerful in some individuals than in others.

Dr James Ritchie said that all present must view with great satisfaction some of the results reported by Dr Brakenridge, and also that most interesting case exhibited and described by Dr Affleck. These practical results were most encouraging. As to the pathology, he believed, with Dr Russell and Dr James, that the disease is due to an increased destruction of blood, and not to an error in the blood-forming organs. He would not attempt to cover the ground which had been so ably occupied by Dr Russell, but he wished to give three further sets of facts which led him to take the view of the pathology which he did. *First*, clinically there was a class of cases of anæmia in which one could find no fault with the kind and quantity of food, the indigestion was not of such long duration as to account for the anæmia which existed, and there was no history of loss of blood or of wasting discharge. In addition to pallor the complexion was sallow, grayish, the urine was high coloured, and contained a very evident quantity of albumen, and in the conical glass with nitric acid showed the upper band of haze described by Dr Steven as characteristic of liver disorder. Treatment directed to the stomach produced no marked benefit, but treatment directed to the liver was followed by disappearance of the symptoms and of the anæmia. The *second* class of cases had been already alluded to by Dr Smart, viz., those in which decomposition of the intestinal contents was accompanied by anæmia. Restoration of the intestinal contents to a normal condition was followed by improvement in the quality of the blood. The *third* series of cases was of special interest to Dr Ritchie, and was somewhat exceptional. It was a matter of common experience that after birth, in consequence of the altered circulation

through the liver, the newly-born child was more or less jaundiced, and in some cases there was a degree of anæmia. In the speaker's experience of eight children born to a healthy father and mother, although they were all plump, healthy-looking children, of good colour when born, the whole of them became jaundiced to an unusual degree; the anæmia was very alarming, and although four of them had recovered and lived, the other four had succumbed to the anæmia. Two of those who lived were hardly expected to survive, the anæmia and consequent debility were so profound. There was no hæmorrhage on the surface, except in one case in which severe umbilical hæmorrhage occurred, but in addition to the pallor there was a marked lemon tint. The urine was high coloured. The liver and kidneys of one case were examined; they became black at once when treated with sulphide of ammonium, and under the microscope abundance of iron pigment granules were seen both in liver cells and in kidneys. In the first class of cases the anæmia seemed to be due to an excessive destruction of blood in the liver, which was readily arrested by treatment of the liver. In the second class the anæmia was probably induced by the action of ptomaines or some poison from the intestinal tract, acting through the liver. In the third series there was an exaggeration of a condition which occurred naturally, which was often followed by a slight degree of anæmia,and which was almost invariably recovered from; but in all of these children it was much more profound than usual, and in four of them it was so severe as to cause death. In these cases the symptoms had a marked resemblance to pernicious anæmia; there was evidently an increased destruction of blood. In those who survived there seemed to be no error in the blood-forming organs, and the post-mortem appearances showed that there had been an excessive destruction of blood, and they resembled those found in pernicious anæmia.

Dr Norman Walker had three remarks to make. 1. It was surely a point of great interest that the microcytes which were invariably present before the transfusion were never subsequently observed. It seemed a point in favour of Dr James's claim, that transfusion supplied a direct antidote to the poison which was causing the disease. 2. Would it not be well to stain preparations of the blood, before and after the transfusion, to show the effect of that process on those cells which could only be differentiated from each other by their colour proclivities? 3. In reference to the unfortunate injection of septic material in one of the cases—where time could be taken, of course the fractional method of thorough sterilization in plugged flasks was the ideal method, but when needed for immediate use he wished to press the fact that ordinary tap water was a safer solvent to use than old distilled water, even if it had only stood for one day.

Dr Matheson Cullen wished to make a few remarks on the subject under discussion, and that merely from a biological point of view.

What is the explanation of the improvement brought about by the transfusion of blood? Is that amelioration due to the fact that the new blood cells replace the diseased ones and carry on their work? The answer to this question is of great biological importance. If the new cells can live in the body and perform their functions just as if they were in their parent body, then we have advanced far in the direction of the indefinite prolongation of life. For if we can introduce bodies capable of doing the extremely necessary duties of blood cells, we have reason to suspect that other cells in the body will be replaced in a similar manner, and in any case the possession of an ever vigorous blood and always energetic blood cells cannot but greatly postpone the final dissolution. Analogy, however, would suggest that the cells of transfused blood do not live in their new surroundings. We see this in the case of bone and epithelial cells. These have no very important function to perform, and we might surmise that they would live if planted in another body. This, however, does not obtain, for it is found that such cells for the most part die, and are removed by the cells of the body into which they were engrafted. At most they seem but to form a scaffolding by means of which new tissue is built up, and they seem to have some power in directing the new growth to the formation of a tissue similar to themselves, the bone cells suggesting bone, and the epithelial ones epithelium. Thus one might infer that blood cells are not likely to live in other blood, and this we know to be true in cases where the blood of an animal of one species is transfused into an animal of a different species. With regard to the transfusion of blood from man to man, the question has not been so clearly elucidated. Dr Brakenridge thinks they do live, at least for some time, and he points out that the increase in the number of cells immediately after transfusion generally represented accurately the number of new cells added. On the other hand, as I have stated, analogy would point to an opposite conclusion, and some facts appear to support this. Firstly, the transfusion may not be followed by any immediate increase in the blood cells; in Dr Affleck's case there was even some diminution. Secondly, hæmatinuria not seldom follows transfusion, pointing undoubtedly to destruction of red cells within the body. From all this it would seem that the improvement after transfusion in these cases is due to the curative influence of normal blood serum, or of the substances produced by the normal breaking down of the blood cells. Either or both influences may be at work, but in any case the method is brought into relation with recent interesting experiments. Brown-Sequard and D'Arsonval have shown that the serum and debris obtained by crushing the testicle and the gray matter of the brain have a powerful influence on the bodily economy, and the juices expressed from the thymus and the thyroid gland appear to possess similar properties. To the beneficial

action of this new therapeutical group it would appear that we must ascribe the benefit from blood transfusion. But whether the benefit be from vital action or from mere chemical action, it is needless to point out the extreme biological interest attaching to the whole question.

Mr Cotterill said he should like to add his word of thanks to Dr Brakenridge for his able paper; and he felt all the more bound to do so, as the remarks which had fallen from him and some of the other speakers confirmed the view which Mr Cotterill had formed of the value and safety of this method of operation,—a view which he had impressed so strongly on Mr Duncan as to induce him to give it a trial. From his own experience subsequent to the first operation done in 1883 (during which time he had had a large number of very successful cases), he could speak in terms of the highest commendation of the method of transfusion with phosphate of soda solution; and while he quite agreed that the saline solution alone might overcome a temporary difficulty, his experience of it in many cases was that it was decidedly inferior to the transfusion of human blood—the effects of the former being frequently transient and eventually unsatisfactory. As Mr Cotterill felt responsible for the introduction of this method to the Edinburgh school, he could not, in justice to the operation, allow it to be said that it required any great skill on the part of the operator; and while insisting that he in no sense wished to depreciate the character of Mr Duncan's operations, he was confident that one of the greatest advantages of the method was the ease with which the operation might be carried out. Whichever of the two theories of the method of working of the transfused blood eventually turned out to be the correct one, meanwhile the blood in its entirety might be freely and safely injected by this method, if the following points were attended to,—firstly, asepsis; secondly, patience in doing the operation very slowly; and, thirdly, care in keeping the blood which is being injected at a proper temperature.

Mr John Duncan said he quite agreed with Mr Cotterill that the operation required little skill or dexterity. There was more skill required by a carpenter in making a chair than by a surgeon in any operation. But knowledge and care were essential in all, and he could not too strongly insist that while this was an operation easily done, it was one in which a little carelessness might produce disastrous results.

Dr Brakenridge, in reply, thanked the members of the Society for the patient hearing they had accorded him. He said that his paper would have served a useful purpose had it done nothing more than originate the interesting discussion which had followed; for he thought it a good thing that the work being done, and the views held, in the Edinburgh Medical School on this important subject should be thus expressed and published. The purpose of his paper was, as he had stated, *therapeutical* rather than *patho-*

logical; but it appeared to him that the results obtained could not fail to have an important bearing on the interpretation of the pathology of the disease; and he was glad that they had been so accepted by the various speakers who had taken part in the discussion. There was, he admitted, much plausibility in the view taken by Dr James. It was quite in accord with the facts noted in these cases, and it might ultimately prove to be correct. Meanwhile, until further light should be thrown on the subject, he would be content—maintaining a quite open mind—to hold provisionally, that, whatever might be the starting point in the disease —and there might be more than one—the main phenomena were due to imperfect genesis and consequent tendency to easily induced and early death of the blood and blood corpuscles. Rindfleisch has (Virchow's *Archiv,* B. 121) recently adduced strong evidence in favour of this view. He (Dr Brakenridge) wished to emphasize the fact that on the one hand, in all the cases in which there had been no considerable rise of temperature after the transfusion, there had occurred that immediate increase in the number of the corpuscles which he had described; whereas, on the other hand, in all the cases in which the corpuscles had fallen in number shortly after transfusion, the operation had been followed by a marked rise of the temperature, or the fall had been otherwise accounted for. It was probable, he thought, that in those latter cases the corpuscles destroyed were taken from the feeble, diseased ones previously present in the blood, and not from the healthy blood added by transfusion. Assuming this to be the case, the ultimate progressive rise in the number of the corpuscles, which had been noted in almost every case where the feverish paroxysm had at first been followed by a fall in the number of the corpuscles, was brought into harmony with the facts noted in the non-feverish set of cases in which an increase in the number of the corpuscles immediately followed the operation, and afterwards became progressive. However this might be, the main object of his paper had been to demonstrate the safety and success of transfusion of human blood by the method employed in these cases, as a therapeutical measure in the treatment of pernicious anæmia. He hoped that, in the great interest attaching to the still unsolved problem of the pathology of the disease, this would not be lost sight of.

Meeting XII.—July 6, 1892.

Mr Joseph Bell, *President, in the Chair.*

I. Exhibition of Specimen.

Dr Clarkson showed for *Dr James Carmichael* the BRAIN from a child, 2½ years old, who died from abscess of the left lateral lobe

of the cerebellum. The abscess cavity was in size $1\frac{1}{2}$ by $1\frac{1}{4}$ in., and contained thick creamy pus. There was no other morbid appearance of the brain except very limited meningitis at the margin of the internal auditory meatus, over which part the bone was quite bare. The middle ear was filled with unhealthy granulations, which extended into the internal ear, the bony walls of which were carious. The clinical history of the case was that of otorrhœa, which commenced eighteen months before the child's death. The first symptom of gravity showed itself five weeks ago, when facial paralysis was observed. A week before admission to hospital drowsiness came on, accompanied by vomiting of a cerebral type. When brought into hospital the child was found to be remarkably well developed and well nourished. He had well-marked partial paralysis of the left side of the face, the pupils were equal, of medium size, and reacted to light and accommodation. There was no paralysis, contraction, or twisting of any of the muscles of the limbs. On crying there was evidence of slight inspiratory spasm of the glottis. The sensory functions were undisturbed. On examination, the left ear was discharging extremely fœtid pus, the drum membrane was destroyed, and the tympanic cavity filled with granulations. Ophthalmoscopic examination of the eyes showed haziness of the edges of both optic discs, slight obscuration of the vessels, with whitish exudation as they pass over them. The child was otherwise healthy. The temperature maintained an average normal range until shortly before death, when it ran up to 103°·6. The diagnosis was cerebral abscess, probably in the temporo-sphenoidal lobe. In consultation with his colleague, Mr Joseph Bell, it was decided to trephine the skull over the temporo-sphenoidal region. This was accordingly done by Mr Bell. The dura was found to be very tense, and on incision the brain bulged out into the opening. The temporo-sphenoidal lobe was explored in all directions with an aspirating needle, and from one of the punctures a quantity of bloody fluid escaped, relieving the tension completely. On account of the state of the child, it was not considered desirable to trephine in any other situation. The wound was accordingly stitched up, and although he became somewhat more sensible for a time, he gradually lapsed into coma, and died the next day. The case was in its clinical features an ordinary one, but none the less interesting, showing, as usual, an absence of any special localizing symptoms, which is the rule whether the lesion is in the temporo-sphenoidal region or the cerebellar lobes.

II. Exhibition of Instrument.

Dr Alexander James showed a SPHYGMOMETER.

III. Original Communications.

1. ELECTRO-DIAGNOSIS BY MEANS OF THE URINE.

By Dawson Turner, B.A., M.D., F.R.C.P.Edin., M.R.C.P. London ; Physician to the Livingstone Memorial Dispensary ; Lecturer on Medical Electricity at Surgeons' Hall.

A preliminary account of this research was laid before the Royal Society, Edinburgh, in December last. The present paper, based upon a more extended series of experiments, is a further contribution to the same subject.

The object of the inquiry was to ascertain the electrical resistances of various kinds of urine both in a state of health and disease. The measurements were made by means of a Wheatstone's bridge with alternating currents and a telephone, according to Kohlrausch's method, and at a temperature of 65° Fahrenheit. The resistance could also be estimated, though less accurately, by the current from a few Daniel cells, a galvanometer, and an application of Ohm's law. I made some seventy experiments by this method. It would appear from the observations, some 500 in number, that the specific resistance of a normal urine amounts to about 45 ohms, and that it varies as a rule inversely with the specific gravity. The latter is a measure of the amount of solids in solution, and particularly of the urea. It might be supposed, then, that the resistance depends also mainly upon the amount of urea; this, however, is not the case. A number of experiments were made with artificial solutions of urea, sodium chloride, phosphates, sugar, etc., in distilled water (see Tables 1, 2, 3, 4, 5), and from these it is clearly apparent that the electrical resistance depends almost wholly upon the salts, chlorides, phosphates, sulphates, etc., and that it is only when these are absent or diminished that the influence of the urea makes itself felt. The resistance is, therefore, a measure of the chemically active substances in urine, of the salts, and very much less of the inert urea. This gives us a simple and rapid method of estimating the constitution of a urine as regards its salts, while the specific gravity, in the absence of sugar, is a guide to its urea. In a normal urine, as already stated, these go more or less inversely together; given the specific gravity and the resistance can be estimated, and given the resistance and the specific gravity can be calculated.

In diseased conditions there are certain exceptions to this rule,—"That the resistance varies inversely with the specific gravity," which can be arranged accordingly as they occur in acute or chronic diseases. Excluding acute infectious diseases and local surgical affections, the two most prominent exceptions are,—amongst the former, acute croupous pneumonia; amongst the latter, diabetes mellitus (Table 6).

That the urine of a case of pneumonia should offer a higher electrical resistance than would be predicated from its specific

gravity is easily understood, when the great diminution of the chlorides in the urine of a case of this disease is remembered; but the increased resistance of a diabetic urine affords a question of a more interesting nature. In this disease the specific gravity of the urine is high, and the electrical resistance offered by it is also high, and sometimes very high. Thus the specific gravity may be considerably above 1030, and the resistance at the same time as much as 150 ohms, while the resistance of an ordinary urine of a specific gravity of 1030 would probably be below 40 ohms (see Table 6).

Upon what does this increased resistance depend? to a diminution in the salts, or to the presence of the sugar? Almost wholly to the former. The tables Nos. 2 and 4 of experiments with artificial solutions show us that a 1 per cent. solution of sodium chloride has a resistance of 59·5 ohms, and that the addition of 10 per cent. of grape sugar only raises the resistance to 68 ohms; a 2 per cent. solution of sodium chloride has a resistance of 29·25 ohms, the addition of 20 per cent. of sugar raises it to 51 ohms, and of 30 per cent. of sugar to 63·75 ohms. With smaller percentages of sugar there may be scarcely any effect (Table 5).

It cannot, therefore, be doubted that the sugar, probably hindering diffusion by its viscosity, does to a slight extent raise the resistance of a saline solution. (It will lower the resistance of distilled water when by itself.) But by far the greater part of the increased resistance of a diabetic urine is due to the relatively great diminution in the salts, and it is to the merit of the electrical testing that this is brought so prominently forward. It will be found further that this resistance usually diminishes with the amount of sugar passed, and that it may be utilized as a test of the patient's progress. Whenever we find a high specific gravity together with a high electrical resistance (above 80 ohms specific resistance), we may suspect the presence of sugar. The specific gravity is raised by the sugar, and the resistance is increased by the diminution of the salts (Table 6).

In acute and chronic Bright's disease the resistance is high, also usually in chronic bronchitis, phthisis, mitral disease, and anæmia, particularly pernicious anæmia. The effect of diet has to be considered. Copious draughts of water increase the resistance. Taking much salted food diminishes it, etc. The urine passed in the early A.M., if a late dinner or supper is eaten, commonly has a relatively low resistance. Some of the principal tables are appended. No. 1 exhibits how small the effect of urea is compared with that of sodium chloride, Nos. 2, 3, and 4.

TABLES OF THE ELECTRICAL RESISTANCES OF ARTIFICAL URINES.

1. *Urea.*

Strength.	Specific Resistance in Ohms.	Strength.	Specific Resistanc in Ohms.
3 per cent. .	. 382·5	1 per cent. .	. 977·5
2 „ .	. 569·5	0·5 „ .	. 1785

2. *Sodium Chloride.*

Strength.	Specific Resistance in Ohms.
2 per cent.	29·25
1 „	59·5
0·75 „	79·9
0·5 „	110·5

3. *Urea and Sodium Chloride.*

Urea.	Trace.	
3 per cent.	1 per cent.	55·25
2 „	1 „	57·8
1 „	1 „	57·8

4. *Grape Sugar and Sodium Chloride.*

Trace.	Sugar.	
1 per cent.	10 per cent.	68
2 „	20 „	51
2 „	30 „	63·75

5. *Urea and Sodium Chloride and Grape Sugar.*

Urea.	Trace.	Sugar.	Specific Resistance in Ohms.
2 per cent.	1 per cent.	8 per cent.	68
2 „	1 „	4 „	59·5
2 „	1 „	2 „	59·5

6. Table of Specific Electrical Resistances in Diabetic and other Urines.

Number.	Disease.	Sp. gr.	Reaction.	Temp.	Urea, grs. per oz.	Albumen.	Sugar, grs. per oz.	Specific Resistance. Ohms.
1	...	1026	Acid	65°	11	...	...	31·45
2	...	1024½	Acid	„	11·7	...	...	31·45
3	...	1027	Acid	„	10·29	...	...	33·15
4	...	1027	Neut.	„	12·16	...	...	36·55
5	...	1027	Acid	„	13·1	...	...	38·59
6	...	1017	Neut.	„	7·4	...	...	43·86
7	...	1012	Acid	„	4·21	...	...	62·9
8	...	1009	Acid	„	5·85	...	...	73·9
9	...	1010	Acid	„	1·5	...	...	119
10	Exoph. goitre	1005	Acid	„	1·4	...	...	153
11	Diabet. mell.	1034	Acid	„	4·68	...	25	156·4
12	Diabet. mell.	1034	Acid	„	6·08	...	27	139·4
13	Diabet. mell.	1034½	Acid	„	5·6	...	30	131·75
14	Diabet. mell.	1034	Acid	„	6·08	...	27	129·2
15	Diabet. mell.	1040	Acid	„	8·1	...	28	124·44
16	Diabet. mell.	1025	Acid	„	6·08	Trace	14	91·8
17	Diabet. mell.	1036	Acid	„	13·1	...	17	54·4
18	Artificial diabetes.*	1040	Acid	„	10·76	...	30	37·4

* 30 grs. of grape sugar per oz. added to normal urine.

By this means, then, we are enabled to make an immediate rough estimation of the constitution of a urine, and particularly of its salts, chlorides, phosphates, sulphates, etc.,—a result which could only otherwise be arrived at by a tedious chemical analysis. The specific gravity of a urine depends (in the absence of sugar) mainly upon the urea; the electrical resistance upon the salts; by combining these we can at once form a fair idea of a urine's constitution, and of the efficiency of the kidneys. For illustrations I append the resistances of a urine of a patient, J. B., suffering from subacute Bright's disease.

Electrical Resistance of J. B.'s Urine (Subacute Bright).

Date.	Sp. gr.	Urea, grs. per oz.	Albumen, etc.	Specific Resistance.
Dec. 8	1011	4	Blood	114·75
„ 11	1012	4·68	„	106·25
Jan. 9	1013	4·68	Albumen	89·2
„ 20	1011	3·6	Blood	93·5
„ 30	1015	3·74	„	76·5
Feb. 11	1010	3·04	„ (less)	73·1
„ 15	1014	4	„	61·2
„ 23	1015	4·68	„	59·5
March 5	1016	5·6	„	61·2
„ 12	1017	4·68	„	74·8
„ 24	1020	9	Albumen	63·75
May 28	1024	8·19	„ (less)	42·5

When first seen he had been ill about a fortnight, was confined to bed, and had the ordinary symptoms of acute Bright, with the addition of severe abdominal pain. Upon being placed upon suitable diet and treatment he began slowly to amend, but had two relapses. The blood and albumen in the urine gradually became less marked, but traces of the former persisted until March 12. The patient was kept on milk diet until March 15. He has now been at work for two months, and considers himself to be quite well. His urine, however, still contains some albumen. It will be noticed that the resistance fell before the specific gravity or urea had much altered; it was, in my opinion, much the more trustworthy guide. Such illustrations could be multiplied; I will add one other, which is interesting from the circumstances under which the improvement occurred. A patient, L—— (under Dr Affleck's care in the Edinburgh Royal Infirmary), suffering from pernicious anæmia and growing worse, had 5 oz. of blood transfused on February 16. After this he steadily improved. The resistances taken a month before, just before, just after, and some time after, are very instructive.

CASE OF L—— (PERNICIOUS ANÆMIA).

Date.	Sp. gr.	Urea, grs. per oz.	Albumen.	Specific Resistance.
Jan. 16	1017	9·36	...	68
Feb. 11	1015	6·31	...	78·2
„ 16*	1014	7·7	...	115·6
„ 16†	1014	6·5	Blood	115·6
„ 17	1012	6·08	...	114·75
March 1	1020	7·02	...	51
„ 12	1019	7	...	44·2

* Before transfusion. † After transfusion.

It is quite obvious that in this case the resistance best reflected the patient's condition,—steadily increasing as he grew worse, and diminishing as he grew better. Thus it rose before transfusion from 68 to 115·6 ohms, and it fell after transfusion from 115·6 to 44·2 ohms. It has from many other observations appeared to me, that so far as the condition of the urine reflects a patient's condition, one of the best gauges of it is the electrical resistance; and more than that, even in other conditions, when the kidneys were supposed to be quite unaffected, as in the case of pernicious anæmia, the electrical resistance will usually closely follow any changes in the patient's condition, changes sometimes of so slight a nature that they would otherwise almost escape notice. The urine to be examined should be fresh, and should be a specimen taken from the whole amount passed in the twenty-four hours. The resistance diminishes as the urine is kept from fermentation changes. This method of examining the urine is completely in its infancy, and my impression is that much may be expected from its further development. Not that it is intended that it will in any way replace the ordinary methods of examination; it is an adjunct to them, and one apparently of much promise, that can be very rapidly carried out, but whose precise value has yet to be determined. Sir W. Roberts, in his lectures upon gout and the urates of soda, points to the influence of the saline constituents of the fluids of the body. It is probable that this method may prove of advantage in this direction.

In conclusion, I desire to express my great indebtedness to Dr Affleck, both for many of the specimens of urine obtained from patients under his care, and for the encouragement he has continually extended to me in prosecuting this inquiry. My thanks are also due to Dr Milne Murray for advice in arranging some of the electrical details.

Dr James stated that he had listened with great interest to Dr Turner's paper. He confessed that it was a subject about which he knew very little, but he believed that it was a valuable one, and he knew it was one in the working out of which our Continental neighbours were ahead of us. He thought the Society should be

specially thankful to Dr Turner for having brought it forward. What struck him as being of special importance was its value in prognosis. In the cases—notably in the Bright's and pernicious anæmia cases—the gradual return of the specific resistance of the urine to the normal accompanied most closely the recovery of the patient. Now he should like to know if Dr Turner had any theory as to any constituent of the urine on which the specific resistance of the secretions might depend. They knew that, as shown by experiments on animals, the toxicity of the urine differs in different cases in a way which chemistry cannot well explain. Were they to believe that similar differences might also be demonstrated by testing its specific resistance?

Dr Gillespie remarked that the experiments of Dr Dawson Turner were very interesting. He thought, however, that the deductions were rather far-fetched, and that the number of possible combinations and permutations negatived any useful result. Although the day might arrive when such a proceeding was possible, he was afraid that physicians of the present day were not confiding enough to gauge the state and prognosis of their patients by the electrical resistance of their urine. He preferred the ordinary chemical tests both for inorganic and organic salts, and believed that the ureometer could tell with greater accuracy the variations in the daily amount of the urea, and Fehling's test the amount of sugar, than any electro-diagnostic method. He was sorry that Dr Turner had forgotten, in tabulating his results, that analysis of the day's urine only yielded data that could be depended on if the whole quantity passed in the twenty-four hours were taken; deductions from specimens passed at various times of the day were absolutely erroneous.

Dr Turner said he did not feel himself yet in a position to be able to say definitely upon what individual substance in the urine the resistance chiefly depended,—the experiments with artificial urines point to the sodium chloride. The resistance of distilled water was infinite, that of ordinary tap water was about 25,000 ohms. The only standard that one can apparently take is that of the average resistance of normal urine, which amounts to about 45 ohms. The method was not intended to supersede any of the ordinary tests for urine, but to supplement them, and it was as yet quite impossible to say what its value might hereafter turn out to be. The method was a completely new and original one, but it had already shown itself to be one of extraordinary delicacy.

2. THE ANTISEPTIC TREATMENT OF PERNICIOUS ANÆMIA.

By G. A. Gibson, M.D., D.Sc., Secretary R.C.P. Ed., Assistant Physician to the Royal Infirmary, and Lecturer on the Principles and Practice of Medicine at Minto House, Edinburgh.

Being unable to attend the last meeting of the Medico-Chirurgical Society of Edinburgh, it was not in my power to take part, as I had promised, in the discussion on Transfusion in Pernicious Anæmia. The loss was all my own, and it is a matter of great regret to me that I was prevented from hearing the interesting debate which followed Dr Brakenridge's paper. I have endeavoured from the medical press to ascertain the views which were laid before the Society, and I am indebted to the courtesy of the Secretaries for the offer of this opportunity to submit the remarks I had intended to make on the occasion referred to.

My purpose is but a limited one,—to bring before the Society the views of Dr William Hunter, and to state the results which have in my hands followed the adoption of his recommendations.

Briefly stated, the conclusion to which Dr Hunter has been led is that in cases of pernicious anæmia there is excessive hæmolysis. He has arrived at this opinion[1] from observing that in this disease there is a largely increased excretion of urinary pigments, that blood pigment can be detected in cells obviously of renal origin, and that the urine contains a large increase of iron. The symptoms of the disease being so suggestive of something akin to ptomaine poisoning as to lead him to suspect some toxic bodies, analogous to the cadaveric series, to be the cause of the increased blood destruction, he investigated the excretions with the view of ascertaining if they lent any support to this conjecture. From the excretion of aromatic substances he was able to determine that there was no absolute increase in the amount of putrefaction in the alimentary canal, but from the presence of what seemed to be special diamine bodies in the urine he concluded it to be probable that some special organism was the cause of the blood destruction, acting, no doubt, in the alimentary canal.

The practical suggestions which Dr Hunter makes as the result of his elaborate investigations are, that the diet should be as far as practicable fatty and farinaceous, so as to lessen to as great a degree as possible the liability to blood destruction, and that the medicinal treatment should be to combat the tendencies of the disease in the same direction by means of antiseptics,—that which he particularly recommends being β naphthol.

During the latter part of last winter, when, in consequence of

[1] See *The Lancet*, vol. ii. for 1888, pp. 555, 608, and 654; *The Practitioner*, vol. xliii. pp. 161, 321, and 401, 1889; *British Medical Journal*, vol. ii. for 1891, pp. 1 and 81.

the unfortunate illness of Professor Grainger Stewart, his wards were under my care, I had an opportunity of putting Dr Hunter's opinions to the test of experience, and I shall now, without further preface, lay the results before you.

J. C., aged 55, until lately a tea-planter in Northern India, was admitted to Ward XXII. of the Royal Infirmary, 29th January 1892, complaining of weakness and giddiness, with pains in the head and shoulders. The patient had no hereditary tendencies bearing upon the condition then present. While in India he had suffered once from cholera, and he had also had some attacks of malarial fever. Five years ago he suffered from renal calculus on the right side, at which time his urine was, as he says, like claret. The present illness began, about a month before admission, with weakness, giddiness, headache, and palpitation. As these symptoms became progressively worse, he came to Edinburgh for the purpose of placing himself under treatment.

On admission the patient was found to be 5 ft. 6 in. in height; the weight was 11 st. 6 lb. He was well developed and muscular. His complexion was somewhat lemon-tinted, his lips and gums were pale, as were also the conjunctivæ, and he had some œdema about the ankles.

The patient had little appetite, but he had also little thirst. There were no feelings of discomfort during digestion, but there was obstinate constipation. The teeth were good; the tongue was large, pale, flabby, and indented. There was no change in the size of the liver or any of the other abdominal viscera. The fæces were colourless. The pulse was of low tension, moderately filled, regular and equal, its rate being 74 per minute. There was a distinct apex-beat in the fifth interspace, $2\frac{3}{4}$ in. to the left of the mid-sternal line. At the level of the fourth rib the right edge of the heart was $2\frac{1}{4}$ in., and the left edge $3\frac{1}{4}$ in. from mid-sternum. A continuous venous hum was heard over the veins of the neck, and a systolic murmur of very soft character over most of the precordia, with its maximum intensity in the tricuspid area.

On careful examination of the blood with the Thoma-Zeiss hæmacytometer the red corpuscles were found to number 800,000 per cubic millimetre. Changes in size and shape of the red corpuscles were very obvious, but there was no absolute change in the number of the leucocytes. The spleen and lymphatic glands were of normal size. In regard to the respiratory system, there was a history of frequent attacks of epistaxis. There was no change in the fundus oculi. The urine was pale, and no changes of importance were present in it. The temperature was never at any time abnormal.

That this case was one of pernicious anæmia there could be no reasonable doubt, the only characteristic symptoms of that disease which were absent in our patient being retinal hæmorrhages and dark urine.

For the first few days after admission the patient was treated by means of arsenic in various forms, but this had to be discontinued, as the drug produced great gastro-intestinal irritation, showing itself by vomiting and diarrhœa. He was becoming progressively weaker, and the œdema of his feet and legs was increasing; but his blood, frequently examined, remained until 16th February in the same condition, *i.e.*, it contained 800,000 red corpuscles per cubic millimetre.

On 18th February, in addition to the administration of two grains of the protochloride of iron three times a day, the patient was ordered to take small quantities of the perchloride of iron largely diluted with water.

On 2nd March, although the red corpuscles were 920,000 in number, and his weight had reached 11 st. 9 lb., the patient was so reduced in strength that it was clearly necessary to resort to some other means of treatment. The further use of iron was suspended, and it was resolved, with the permission of the patient, which was freely granted, to have recourse to transfusion of blood.

The operation was accordingly performed by Mr Cotterill on the following day,—six ounces of venous blood, carefully defibrinated and diluted with the usual saline solution, being introduced with every precaution into the patient's right arm. There was but little constitutional disturbance in consequence of the transfusion, and on the 4th March the number of red discs had reached 1,120,000. By the 9th March the number had fallen to 1,000,000, and by the 12th March to 920,000. His energy during this time was steadily failing, the œdema was gaining on him, the condition of pulse and heart was becoming worse and worse. The question now before us was whether to have recourse to repeated transfusions or to attempt some other method of treatment. The patient himself was averse to further transfusion, and this fact weighed with us in coming to a decision.

After taking every circumstance of the case into consideration, we resolved to follow the suggestions of Dr Hunter. For two or three days the patient was fed with artificially digested food, and on the 18th March the use of β naphthol was begun, two grains in pill being administered three times a day.

On the 22nd March the number of red discs was 1,000,000, on the 1st April his weight was found to have fallen to 10 st. 10 lb., and on 2nd April the red corpuscles only numbered 800,000. During the three weeks of treatment by means of the β naphthol a good deal of intestinal disturbance had from time to time troubled the patient, and on the 2nd April a severe attack of diarrhœa set in. Whether the drug was the cause or not was uncertain, as at that time there was an epidemic of diarrhœa in the ward; but the attack was severe and lengthy, as in spite of all kinds of medicines the symptom did not abate until a fortnight had nearly passed away. On the 16th April the number of red cor-

puscles was 1,600,000, and the colour of the skin and mucous membranes was greatly better than at any time since the patient's admission; the œdema, moreover, had quite disappeared, but his weight, as might be expected, was only 10 st. 1 lb.

On the 19th April the administration of the β naphthol was resumed, but the patient only took two pills daily.

On the 23rd April the number of red discs was 1,700,000, and on the 2nd May it had reached 1,850,000. The patient had improved in every way—his strength had greatly returned, his appetite was much better, and his appearance had undergone much improvement. It was considered advisable that he should leave the Infirmary and live in the country, where he might enjoy fresh air; he accordingly took up his abode in the vicinity of Edinburgh on 4th May.

On the 20th May the red corpuscles were 2,000,000, and on the 26th they amounted to 2,080,000. His weight was 10 st. 8 lb., and his strength much greater, so that he was able to take short walks without experiencing much fatigue. He was now permitted to return to his home in Galloway, and was enjoined to continue the use of the β naphthol, to which was added protochloride of iron in doses of two grains twice a day.

On the 18th June the blood was examined by Dr M'Lelland of Wigtown with Gowers' hæmacytometer, and found to contain 1,840,000 red discs. Although there was thus a diminution in the number of the corpuscles, the patient was still improving in strength and energy.

On the 3rd July his weight had risen to 12 st. 1 lb., the number of the red corpuscles was 2,320,000, and the patient was able, without feeling fatigued, to walk more than four miles.

There are many points in this case somewhat difficult of solution.

It will, no doubt, be suggested that the considerable improvement obtained may have been due to the transfusion of blood. This was, however, followed by such a long period unattended by any gain that it seems to me such a suggestion must be dismissed.

One matter of great interest is that during the period of diarrhœa the red corpuscles were doubled. No doubt the drain of fluid from the patient's alimentary tract would not only remove œdema, but would also relatively increase the red corpuscles. It may possibly be held that the doubling of the corpuscles was simply caused in this way, but it is hardly possible that so noteworthy a change in their number could be thus produced; and I am much more inclined to believe that the explanation must be sought in an antiseptic action on the alimentary canal, coincident with the diarrhœa.

What is more difficult to explain is the falling off in the number of the red discs during the first period of administration of the β naphthol, and the later diminution after the patient's return to his home.

It was carefully considered whether there might not be some malarious influence in the case, and the possibility must be granted. No evidence of parasitic causation could be ascertained.

Such are the facts as to this case which I desire to lay before the Society. They are imperfect in many ways, and more especially in not containing any estimation of the hæmoglobin. This omission is due to the epidemic of influenza, which caused so many changes amongst the Resident Physicians as seriously to interfere with the carrying out of continuous observations.

In order to have a control observation as to the effect of antiseptic substances in cases of anæmia which are probably not progressive and pernicious, I have administered β naphthol to a considerable number of patients. So far, there has been in every instance a noteworthy increase in the number of the coloured corpuscles under the influence solely, as far as I am able to judge, of such agents.

One of these cases may be briefly referred to as an example of the effects produced by β naphthol.

K. S., aged 20, unmarried, and employed as a kitchen-maid, came under my observation on 6th May 1892, complaining of breathlessness and headache.

The patient was pale, and her mucous membranes were blanched, but there was no œdema, and the catamenia had undergone no change. On physical examination of the circulatory apparatus, the pulse was found to be of moderate tension; the heart somewhat dilated, the right border extending to 2 inches from the middle line, and the left to $3\frac{1}{2}$ inches in the opposite direction; there was a distinct venous hum in the veins of the neck; and a soft systolic murmur was audible in the tricuspid and pulmonary areas of the precordia. The blood examined by means of the Thoma-Zeiss hæmacytometer, was found to contain 1,950,000 coloured corpuscles per cubic millimetre; some of these were slightly altered in outline, but neither megalocytes nor microcytes were present.

The case appeared to be one of simple anæmia, and was selected in order to ascertain what effect the exhibition of intestinal antiseptics would have upon the state of the blood. The drug administered was β naphthol, two grains of which were given in pill form three times a day after food, and no alteration was made on the food, surroundings, or occupation of the patient.

The number of the coloured corpuscles, as determined from time to time, may be stated in tabular form, as follows:—

6th	May	1892	1,950,000
13th	„	„	2,450,000
20th	„	„	2,675,000
27th	„	„	2,550,000
11th	June	„	2,850,000
2nd	July	„	3,200,000

In this table is shown a considerable increase, interrupted once by a diminution. This fall in the number of the coloured corpuscles was apparently caused by the occurrence of the catamenial period, which had taken place a few days before the examination of the blood on the 27th May. The general result of treatment by means of an antiseptic substance in the case of an apparently simple anæmia is therefore similar to that observed in regard to pernicious anæmia.

It was not my intention to do more on the present occasion than to lay these facts before the Medico-Chirurgical Society, but it is difficult to abstain from drawing one or two provisional conclusions. The results to some extent support the explanation given by Bunge of the *modus operandi* of iron in the cure of anæmia. I am not sure whether his views have been previously mentioned in the Society or not, and it may be allowed me in a few words to state them. We have the authority of Schmiedeberg for believing that iron, except in organic combination, is not absorbed from the alimentary tract, and, stated succinctly, the explanation advanced by Bunge of the unquestionable effects of iron in almost every form of anæmia is, that such iron as is given medicinally seizes certain unknown poisons formed in the alimentary canal, and prevents them from acting upon the iron contained in the food, and rendering it inert. It seems to me that this theory—which, however, I am informed by my friend Dr Stockman is in some respects not based upon unassailable grounds—receives some support from the results obtained by the use of a substance like β naphthol. And the fact that the continuous use of saline purgatives may in itself, but still better along with iron, remove anæmia—a fact lately urged by Sir Andrew Clark—lends itself to the support of Bunge's view.

In the simple case described above iron would, no doubt, have more quickly cured the patient, but the point I desire to urge is that by antiseptics anæmia may be remedied. In a large number of cases treated by me in hospital work and in private practice, I have found extremely rapid improvement to follow the administration of iron along with β naphthol.

I would finally mention to the Society that in order to obviate the irritation sometimes caused by the β naphthol, I have found the best means of administration to consist in combining it with some bismuth preparation, such as the salicylate.

It is a pleasant duty to express my obligations to Mr Cotterill for his kindness in performing the operation of transfusion in the case of pernicious anæmia which has been narrated, to acknowledge the zealous devotion shown by Drs John Spurway, Stodart Walker, and Cuthbert Thompson in carrying out the details of observation in both cases, and to tender my thanks to Dr M'Lelland for his courtesy in keeping me informed as to the progress of the patient.

Additional Remark.

Since the foregoing observations were laid before the Society, Dr M'Lelland has reported a continued improvement in the condition of his patient. By 3rd August the number of coloured corpuscles had reached 2,830,000 per cubic millimetre, and the weight had risen to 12 st. 5 lb., and on 3rd September they were respectively 3,620,000 and 12 st. 3½ lb.

Surgeon-Major Black said, with respect to the nature of the case related by Dr Gibson, he would venture to suggest that it contained some of the elements of scurvy, and might be treated with benefit by increased use of sugar in the diet, and administration of lime juice, as antiseptics in action on the intestinal canal and blood.

Dr Gillespie looked upon Dr Gibson's first case as a possible example of cure by transfusion, the second might have done as well from the mere rest from work. With regard to the causation of pernicious anæmia, especially with regard to the marvellous results of transfusion, he was inclined to look for the origin of the disease in the absence of some body allied to the class of ferments. Six ounces of blood injected into a body containing ten pounds of blood, however healthy the first and impoverished the second might be, could not conceivably cause a continuous improvement unless some body requisite for the proper formation of healthy blood corpuscles was contained in it. An analogous instance might be found in the injection of thyroid extract in myxœdema. The bone marrow might well produce some substance, or contain some substance, which had an influence on the proper production of red blood corpuscles—which are highly specialised cells, and incapable of independent existence. If this substance were at fault the number of weak red corpuscles destroyed during their passage through the portal circulation would be increased, the liver functions overtaxed and deranged, and *propter hoc* digestion in the intestines put out of order. Under such circumstances the absorption from the intestines of alkaloids and ptomaines would be increased, and more corpuscles destroyed as a consequence, not as a cause. Then with the transfused blood an agent was injected which aided in the formation of stronger hæmocytes for a time, and allowed the iron or arsenic a chance of doing good. How otherwise could one explain the good effect of transfusion if after, as well as before the operation the state of the bowels—the cause of the disease according to some—was identical?

Dr Church said—In view of the statement by physiologists that iron cannot be taken into the system except in the form of an organic compound, the speaker inquired whether in Dr Gibson's case of pernicious anæmia iron had been taken after the operation of transfusion. For was it not possible, that by the transfusion of

blood or serum the tissues associated with the process of digestion would be so enriched as to render possible the formation of a small quantity of an organic iron compound of such a nature as could be assimilated by the blood system? In many cases of pernicious anæmia, unaided by transfusion, we knew iron could not be assimilated at all. And if iron were given after transfusion, what share in the cure could be ascribed to the transfusions *per se*, and what to their power of bringing to the iron the necessary elements for the formation of organic compounds with it, and thus to what extent would the iron be rendered an available factor in the treatment? Was the transfusion of blood of real value without at the same time the administration of iron or arsenic?

Dr W. Russell thanked Dr Gibson for bringing his paper before them, as he had been unable to do so on the preceding meeting night, when the discussion on Pernicious Anæmia took place. This paper completed the discussion of this important subject for the Transactions. He thought there could be no doubt that Dr Gibson's case was as certainly a true case of pernicious anæmia as any case could yet be said to be. The effect of the β naphthol was interesting; but seeing the corpuscular richness of the blood did not rise above 50 per cent., the result could not be considered as a cure, and was not equal to the results in the good transfusion cases. Notwithstanding this inferiority in the result in this single case, there was ground for hope that it might be better in other cases, and the naphthol would certainly require to be placed amongst our resources for treatment. The value of Dr Gibson's paper had been greatly enhanced by the observations made on chlorosis with the same substance. Dr Gibson's results did not materially modify his views as to the probable nature of the disease expressed at the last meeting of the Society.

Dr Keppie Paterson referred to the use of β naphthol in typhoid fever, having prescribed it in the case of children. He used it in larger doses than Dr Gibson had recommended for adults, and with no ill effects. In a case which showed serious symptoms early in the fever, there was decided improvement after its administration, the diarrhœa being checked, the temperature diminished, and the nervous symptoms abated. A serious illness changed into one of mild type, and at a stage of the disease when one would have expected intestinal sepsis to be present in greater degree.

Drs Strachan, *Foulis*, and *James* also spoke.

Dr Gibson expressed his thanks to the members of the Society for their kindly reception of his remarks and his interest in the typical case described by Dr Strachan and Dr Foulis. He admitted the justice of Dr Gillespie's suggestion, that perhaps the transfusion had something to do with the improvement which the patient had undergone, but, as he had pointed out in his paper, an interval had elapsed of such length as to render the idea highly improbable. He fully concurred in most of the observations made by Dr

Russell, and with regard to the suggestion of Dr James, he might state that he had once, some years ago, tried perchloride of mercury in a case of idiopathic progressive anæmia without any benefit. In reply to Dr Church he had to state that iron was not administered to the patient in question from the date of the transfusion, *i.e.*, 3rd March, until the end of May, when the protochloride was added to the β naphthol. The remarks made by Dr Foulis led him to fear that he had not made his meaning quite clear. The first part of his contribution was devoted to the treatment of pernicious anæmia, and the second part, detailing the effect of β naphthol in simple anæmia, was, as had been stated, only a check observation. He had no intention of recommending the drug in such cases, and had expressly stated that the experience of the case referred to showed that iron would have effected better results in the same length of time. He had further mentioned that he had obtained excellent results from the administration of protochloride in combination with antiseptic substances. As to the use of β naphthol in enteric fever, as referred to by Dr Keppie Paterson, he had no personal experience, but he had read some time ago with great interest the little *brochure* on the subject by Dr Yeo.

3. SOME DEDUCTIONS FROM A STUDY OF THE DEVELOPMENT OF THE HEART.

By G. A. Gibson, M.D., D.Sc., Secretary R.C.P. Ed., Assistant Physician to the Royal Infirmary, and Lecturer on the Principles and Practice of Medicine at Minto House, Edinburgh, and A. L. Gillespie, M.D., F.R.C.P. Ed., Medical Registrar, Royal Infirmary, Edinburgh.

A consideration of the physical problems arising out of cases in which, on account of the retention of certain embryonic arrangements, deviations from the normal relative pressures existing in the systemic and pulmonic vessels are present during adult life, has led us to seek some means by which it might be possible to establish a basis upon which we may rest with certainty in reasoning upon such conditions. As it seems probable that in the anatomical relations of the two sides of the heart during fœtal life such a secure foundation may be obtained, we have examined the heart of the embryo in order to ascertain the anatomical conditions existing at different stages of its development. During fœtal life the pressure on both sides of the heart must be nearly equal, on account of the free communication allowed by means of the ductus arteriosus and foramen ovale; and it might, *à priori*, be expected that the walls of the ventricles on the two sides of the heart should be of approximately equal thickness.

This expectation we found to be in great part justified by our results, as will be seen in the sequel.

Before entering upon the observations which we wish to lay before the Society, it may be of use to sum up, as briefly as possible, the course of events in the development of the central vascular apparatus.

The vascular system makes its first appearance in the form of two simple lateral tubes in the anterior part of the embryo. These tubes occupy spaces which are continuous with the pleuro-peritoneum, and are composed of two layers apparently developed respectively from the mesoblast and hypoblast. Each tube is continuous in front with the cephalic mesoblast, and terminates behind in the omphalo-meseraic or vitelline veins.

These lateral tubes are developed in the part of the embryo which, on the folding downwards of the body-walls, becomes the ventral wall of the pharynx; and when this wall is completed they meet in the middle line and become fused into a single tube. This remains connected with the vitelline veins behind, and bifurcates in front, giving rise to the primitive aortæ.

At this stage in the development of the vascular apparatus the single median tube begins to pulsate rhythmically, while as yet no muscular tissue can be discerned. This is a point which cannot be denied a considerable degree of significance.

The central vascular tube, at a slightly later stage, becomes somewhat curved by bending over to the right; and at the same time its posterior extremity, connected as we have seen with the venous channels behind, assumes a position dorsal to the rest of the tube. At this period of development superficial constrictions appear upon the tube, dividing it into the venous sinus, the auricle, the ventricle, and the aortic bulb. While these changes are going on muscular fibres are for the first time seen in the substance of the vascular apparatus.

The venous sinus receives the posterior veins already mentioned, and the primitive veins from the cephalic region. It at first communicates freely with the auricle, but afterwards is guarded by venous valves.

The common auricle continues to develop behind the curved ventricular portion of the tube, and in time the right venous valve forms the Eustachian and Thebesian valves, while the left venous valve disappears.

The common ventricle, or curved ventral portion of the tube, receives the blood from the common auricle at its left end, and terminates at its right end in the aortic bulb. It contains in its early stages a fine spongy substance. A slight constriction on its outer surface marks the future separation into right and left ventricles, and a septum, composed of muscular tissue, begins to grow from below and behind, upwards and forwards. This is completed at a later stage by the development of a fibrous septum, which grows from above and before, downwards and backwards. At the same time the spongy tissue in the cavity of the ventricular portion of the apparatus begins to be gathered together in distinct columnar masses.

The common auricle is gradually raised upwards and forwards, so that the opening into the ventricular portion assumes a position

over the inter-ventricular septum, which grows up, and with the assistance of flaps developed from the inner layer—the future auriculo-ventricular valves—divides the orifice into two divisions. A septum is developed in the common auricle from above and behind, downwards and forwards, in which the foramen ovale is formed. Before this inter-auricular septum is complete, the pulmonary vein is developed.

While these changes are proceeding, the aggregation of the spongy tissue of the ventricles into distinct masses goes on, and by this means the columnæ carneæ as well as the musculi papillares with their chordæ tendineæ are formed, the latter becoming attached to the flaps developed at the auriculo-ventricular orifices. The inter-ventricular septum grows up into the aortic bulb and separates it into two divisions, *i.e.*, aorta and pulmonary artery, each communicating with its respective ventricle. Folds of the lining membrane grow up at the junction of the bulb and the vessels, and by their division into segments form the semilunar valves.

To the development of the great arterial trunks it is not necessary to devote much attention. From the aortic bulb two great arterial arches spring, each running forwards, outwards, and backwards, dorsal to the primitive heart, to form the primitive aorta of its own side. The two primitive aortæ unite at an early period in the middle line about the dorsal region. To these arterial arches four other lateral pairs are in succession added, forming five arterial arches on each side. Of these arches the first and second become the external carotid artery and its branches; the third forms the internal carotid artery. The fourth, on the right side, becomes the subclavian artery, and on the left side it forms the arch of the aorta. The fifth, on the right side, develops as the right pulmonary artery, and its distal portion disappears; on the left side it forms the left pulmonary artery, and the portion beyond remains during fœtal life as the ductus arteriosus. The right descending primitive aorta entirely disappears in its anterior part; the left remains as the permanent descending aorta, and joins the posterior part of the right primitive aorta behind, to form the posterior portion of the permanent descending aorta.

From this description of the development of the heart and great vessels it may readily be understood why it is that the pressure on both sides of the heart is very similar, probably being somewhat greater in early fœtal life on the right side than on the left.

We determined to examine the hearts of the human fœtus at different ages, so as to obtain the evidence afforded by the relative thickness of the walls of the ventricles.

The method which we have followed in our investigations has been to make a series of sections through the ventricular portion of the heart of the fœtus at right angles to its axis, and measure the thickness of the walls at different points in the circumference

Fig. 1.

Fig. 2.

Fig. 3.

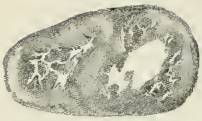


Fig. 4.

Fig. 5.

Fig. 6.

Fig. 7.

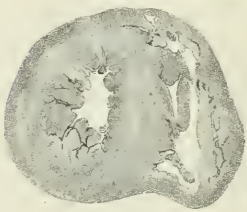
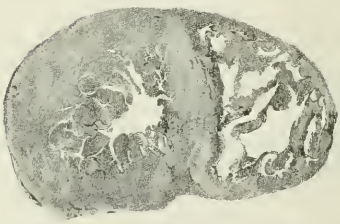


Fig. 8.



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of the ventricles, as well as at different distances from the auriculo-ventricular groove.

The following is a summary of the results:—

	Wall of Right Ventricle.	Wall of Left Ventricle.
1. Fœtus, 3½ months, . . .	1·5 mm.	1 mm.
2. Fœtus, 4 months, . . .	1·5 „	0·5 „
3. Fœtus, 6 months, . . .	4 „	3 „
4. Fœtus, full term; never breathed,	4 „	3 „
5. Fœtus, full term; never breathed,	5 „	5 „
6. Fœtus, full term; lived 24 hours,	3 „	4 „
7. Female infant, 11 months, .	2 „	5 „
8. Male infant, 22 months, . .	2 „	8 „

These figures show that the left ventricle progressively increases in thickness from the third month, while the right ventricle increases up to the hour of birth, and then rapidly diminishes; the thickness of its wall nearly two years afterwards being actually smaller than in the sixth month of fœtal life.

The accompanying Plate gives copies of life-sized photographs of the sections referred to in the above summary, and the numbers correspond to those of the text. The photographs were all obtained looking down on the sections from above, and they were placed with the anterior side above, so that the side of each to the right is the right ventricle.

From these facts we may fairly conclude that the thickness of the walls of the cardiac chambers is exactly proportional to the work which each has to do. And although an inquiry into the teachings of comparative anatomy is not within the scope of this contribution, we may add that the facts which we have laid before the Society as to the condition of the walls are in every respect supported by observations which we have made on the fœtal heart of the horse, cow, sheep, pig, dog, and cat.

The consideration of these points in vascular development is by no means a matter only of scientific interest; on the contrary, the facts which have been briefly described in the preceding pages show how different malformations are possible through arrested or perverted development, and they point to further important truths. The pulsation of the primitive vascular tube, for instance, before the growth of any muscular fibres in its substance, appears to be evidence in favour of the existence of an inherent tendency to rhythmic propulsive movements in the vascular mechanism. And as during fœtal life the blood-pressure must be nearly the same on both sides of the heart, it is undoubtedly a circumstance of real importance, that up to the time of birth the thickness of the walls of the two ventricles is nearly equal on the two sides. This fact may be taken to prove that the amount of strength developed is strictly proportional to the work that has to be done.

Such results lead to far-reaching conclusions in regard to the possibility of the heart adapting itself to widely different morbid conditions. Some further results relating to the proportional thickness of the adult ventricular walls in different diseases have been obtained, but these must form the basis of another communication at some future time.

4. THE HISTORY, CAUSATION, AND PREVENTION OF THE ENTERIC FEVER OF INDIA.

By Surgeon-Captain C. H. Bedford, D.Sc., M.D., C.M. Edin., M.R.C.S. Eng., Bengal Medical Service.

In the following paper I desire to draw the attention of the Society to the subject of Indian Enteric Fever, considered from the hygienist's point of view. I had hoped to enter on the very interesting questions of the pathology, symptomatology, and therapeusis of this condition, but find that will best be considered on a subsequent occasion, as, if considered now, it would increase the size of this paper to very considerable dimensions. I have not considered the question with reference to the prevalence of the disease in camps and on field-service, as this has been exhaustively treated of by Surgeon-Major Duncan, of the Bengal Service, in his splendid work on the subject.[1]

Historical.

Up till 1853, the general impression among members of the profession in India and elsewhere was that no such disease as the "Parisian fever" of Louis, "dothinéntérie" of Bretonneau, or the "typhoid fever" of Stewart and Jenner, existed in the tropics. The writers on Indian diseases anterior to the publication of the classical works of Annesley, Martin, Twining, and Clark, were chiefly naval surgeons, of whom Johnson heads the list. These writers were not in a position to acquire any considerable experience of the disease, for their sphere of observation was limited to the port where their ships were lying, and perhaps to the country in its immediate neighbourhood. They noted cases of "fever with bowel complication," many of which, in all probability, were true cases of enteric fever. Dr John Clark described the fevers which occurred in the Honourable East India Company's ships from 1770–85.[2] He describes an epidemic on the voyage from India, and states that "in the beginning of this fever, inflammatory symptoms chiefly prevailed, frequently with bilious vomiting, but in its progress it changed into a typhus, or was succeeded by a flux." It is not too much to suppose that the group of cases

[1] *Prevention of Disease in Tropical Campaigns.* J. & A. Churchill, 1888.

[2] *Observations on Diseases which prevail on Long Voyages to Hot Countries particularly to the East Indies*, 1792, vol. ii. p. 464.

"which changed into a typhus" were cases of enteric fever. It is not at all probable that remittent fever of such a severe type would persist under the healthful and malaria-destroying influences of a sea voyage. Besides, it is now well recognised that the disease described by various old writers as "ship-fever" was undoubtedly enteric fever. On the other hand, it is probable that a section of Clark's cases were malarial, for the change of diet and air, as well as the chills and nervous disturbances consequent on a sea voyage, frequently induce an attack of ague (in one or other of its many manifestations) in a subject "saturated with malaria;" moreover, malarial diarrhœa would be very likely to appear and persist in such cases, on account of the scorbutic condition formerly so general on long sea voyages. The bilious vomiting noted might have occurred in either enteric or malarial fever in the early stage. That the disease was true *typhus* fever is most improbable. In 1784, seven ships, stationed at the mouth of the River Hooghly, lost 170 men, and the fevers which prevailed were "all attended with diseased viscera.[1] Mr Lyons argues, in the *Indian Annals of Medical Science* (No. 28 for 1871, p. 254), that Charles Curtis, surgeon of the "Medea" frigate, described true enteric fever in India under the designation of "bilious fever and flux." The general teaching of the old school of Indian physicians was to describe fevers with reference to the seasons,—"ardent fever" in the hot weather, "bilious fever" in the rains, and "congestive fever" in the cold season.[2] The first and last were called "continued fevers;" and "bilious fever" was termed "remittent fever," although all three were presumed to have the same cause.

In Sir J. Annesley's works (1828 and 1841 editions) it is noted that, in continued fevers, there occurred "marks of disease in the small and large intestine" which were "confined to their inner tunics . . . . . and that especially the duodenum and termination of the ileum are very frequently diseased in their mucous surface, which is inflamed in patches . . . . . and studded with small ulcerations, particularly the termination of the ileum. . . . . In several cases the ulcerations, which are sometimes large and far apart, at other times small and agglomerated, have nearly penetrated the tunics of the intestine, and in a very few cases we have observed the occurrence actually to have supervened, the contents of the bowel being partly effused into the peritoneal cavity, and having produced peritonitis." Dr Twining, of the Indian Service, who wrote on the fevers of Bengal in 1835, was well acquainted with true enteric fever as the "typhus with abdominal complications" of Richard Bright, or Bretonneau's "dothinéntérie." Yet he asserted that "typhus is rare in India;" and, though familiar with intestinal ulceration in the "congestive fevers of the cold season," regarded it as an accidental concomitant. He notes the "invidious

[1] Chevers, *Diseases of India*, 1886.
[2] Sir W. Moore, *Manual of Diseases of India*, 2nd edition.

invasion, obscure symptoms, slow progress, and protracted course, attended with prolonged stupor and delirium," and thinks that the organic changes in the latter stages of this fever "might establish a resemblance to some cases of European typhus, although the resemblance be not strictly correct in all its details." Sir Ronald Martin, of the Bengal Service, describes a condition which he terms "congestive continued fever of the cold season in Bengal," noting its insidiousness; frequency; the fact that even the most careful Europeans are attacked, and of all ages and sexes, but men especially; the very gradual onset; the fulness of the belly; intense headache and delirium. He ascribes the cause to "exhaustion of the liver by the hot season and rains," and notes that the intestinal mucosa is inflamed and ulcerated. The patients generally recovered if no severe abdominal complications occurred, but when a "generally typhous condition" supervened there was "great peril." He treated his cases by bleeding, calomel, pulv. Jacobi, followed by active salines, and ordered "total abstinence from food, and cool drinks." Barbarous though this treatment seems to us to be, it is interesting to note that two of the most recently proposed drugs for the treatment of this disease are calomel and tartar emetic.[1] Prof. Maclean, I.M.S., late of Netley, saw true enteric fever, in 1838, in Madras, Secunderabad, and in China.

Dr Morehead, I.M.S., writing in 1843, remarks that "a state of ulceration of the glands of Peyer at the end of the ileum also occurs in cases of typhoid remittent with gastro-enteric symptoms, just as it occurs in European continued fever with typhoid symptoms, and similar bowel complications." In this writer's first edition, he utterly denied the existence of enteric fever in India, being accustomed to classify cases of fever *plus* Peyerian ulceration as remittent fever. In the second edition of his work, he steadily maintains that the malady was long of rare occurrence in India. According to Sir W. Moore, Allan Webb of Simla was the first to mention typhoid fever as existing in India, but he probably referred

[1] "The gland lesion appears to be modified by the administration of mercury. Combined with chalk or bicarbonate of soda, calomel in grs. v.–viii. doses, has been found to have a remarkably sedative action on the abdominal viscera, relieving the biliary disturbance which so frequently occurs during the first few days of the disease." It is, of course, contra-indicated in sharp diarrhœa. The lowering of arterial tension, by reducing the amount of uric acid in the blood (Haig), makes it also a vascular sedative. De Simone (in *Rif. Med.*, Dec. 12th, 1891) recommends calomel as an intestinal disinfectant *after* the tenth day of the disease.

Brig.-Surg.-Lt.-Col. Lawrie, Bengal Service, has recently had remarkably good results with doses of $\frac{1}{10}$ gr. up to $\frac{1}{6}$ gr. of tartarated antimony, in ʒj. of water, every second hour till gr. i. in the 24 hours has been taken. The solution should be freshly prepared every day, and its administration continued till the temperature is normal. *No depression occurs from it.* It relieves abdominal pain, tympanites, checks diarrhœa, and prevents cerebral symptoms. It is said to check the inflammatory change in the gut, and to shorten the disease. This remedy is now being extensively tried in India.

to the typhoid *state* when he wrote in 1842; as also did Kirk[1] who, in 1848, wrote: "At the termination of the monsoon (rainy season) and on the beginning of the hot season, congestive enteric fever is abundant all over the country." Here are described, however, the periods of prevalence in a way more exact than any former writer had done.

But it was reserved for Assistant-Surgeon Scriven, of the Bengal Service, to create a very distinct epoch in the Indian history of disease, by the publication of an account of three cases observed by him in Burmah in 1851. To him exclusively belongs the honour of identifying the enteric fever of India with that described by Dr A. P. Stewart in the pages of the *Edinburgh Medical Journal* for 1840 (p. 289 *et seq.*); and later by Sir W. Jenner, in 1846, as the result of his London Fever Hospital experiences. In a later paper, in 1857, Scriven described in detail seven more cases, two of which only recovered, the others dying about the end of the third week, and displaying, at the autopsies, well-marked Peyerian sloughs and ulcers, inflamed solitary glands, and splenic and pulmonary congestion. In 1855, Deputy-Surgeon-General Ewart, of the Bengal Service, first described enteric fever among the *native* prisoners in the Ajmere Jail in Rajputana,—a discovery made quite independently of Scriven's observations of the disease in Europeans in India.

These observations were later supplemented by those of Goodeve, Lyons, Moffat, Massey, O'Brien, Barclay, Cleghorn, Greene, and others of the Bengal Service; and, in the Bombay Presidency, by Surgeon-General Sir James A. Hanbury, K.C.B., then Surgeon of the 33rd Foot, at Deesa, in 1859, and by Peet in Bombay City; and, in the Madras Presidency, by Surgeon-General Cornish, Ranking, Gordon, and Murray.

In 1861, enteric fever first appears in the official returns, and from this period till the present it has never been absent, though the amount of it fluctuates considerably from year to year, as will be shown later. Its admission to the returns was mainly due to a paper written by that very eminent statist, Dr Bryden, then Secretary to the Surgeon-General of the Indian Forces.[2]

In 1869, Surgeon-General de Renzy, Bengal Service, noted its prevalence among native prisoners in the latter half of the year, having first appeared in villages adjacent to the jails. Its severity did not vary concomitantly with that of the famine then prevailing, thus distinguishing it from relapsing fever. It affected more the native women in the villages than the men, because they spent more time in the house, and got larger doses of the poison from the contaminated air and water in the dwellings' vicinity.

In 1880, Deputy Surgeon-General Pinkerton, of Bombay, states that enteric fever had existed there for over fifty years, under the

[1] *Trans. Med. and Phys. Soc.*, Calcutta, vol. xvii.

[2] *On Age and Length of Service as affecting the Sickness, Mortality, and Invaliding of the European Army in India.*

designation of "twenty-one days' bad Bombay fever;" and that among the natives it causes a large mortality all over the Bombay Presidency under the names of "fever" and "remittent fever." On the other hand, we find Brigade-Surgeon Curran—who, it is stated, "made Indian fevers a special study"—reporting that during the fifteen years he was in charge of European military hospitals and large stations in nearly every part of India, he only saw *one* true case of enteric fever, and that was at Murree, a hill station in the Punjab!

Prof. Chevers, Bengal Service, stated[1] that, in 1866, the splendid pathological museum at Calcutta only contained nine preparations of enteric fever lesions—two given by Scriven, one by Eatwell, and three by himself; and he goes on to say, "Thus 120 beds in the largest hospital in India, from 1858 to 1876, only gave nine cases." But I would point out that many cases of enteric fever probably recovered, having been treated all through as remittent fever of a typhoidal or, perhaps, malignant type; that many cases of true enteric fever might have died and were not dissected, in deference to the wishes of the deceased's relatives; and that relatively fewer cases occur in Lower Bengal (in which province Calcutta is situated) than in the other provinces of the Bengal Presidency; and, finally, that many medical men in India (as elsewhere) do not care to dissect a case "post-mortem" which has *appeared* to them to be clearly a case of remittent fever; and that, even if the cadaver was dissected, and typical lesions were found, it would not at all follow that they would be sent to one of the medical colleges' museums, but would be probably buried with the rest of the patient's body. Chevers goes on to express his wonder that the disease should so long have existed in India without having been discovered by the very able men practising their profession there. But the same applies to the many famous European physicians who flourished before, and contemporary with, Stewart and Jenner.

In the beginning of 1889, specimens of spleen and mesenteric glands from two typical cases of Indian enteric fever were sent to Prof. Bernhard Fischer, of Kiel, an eminent bacteriologist, who found the specific bacillus of European enteric fever in *all* the specimens sent. Now, this proves the identity of the Indian and European disease entities in at least some cases, for the general consensus of bacteriological and pathological opinion recognises the causal connexion between Eberth's bacillus and enteric fever. Surgeon-Major Ranking, Bengal Service, in 1889, isolated a bacillus from the (albuminous) urine of a typhoid patient, which, when cultivated on glycerine agar-agar and potato, showed the characteristics of Eberth's bacillus. We must now briefly consider the degree of *prevalence of the disease in India.*

Bryden first drew attention to the great amount of sickness and mortality occasioned by this disease, and pointed out the significant fact that the ratios of "fever" mortality (in the gross) of past

[1] *Diseases of India*, 1886.

years, if taken in relation to months and newly-arrived regiments, were nearly absolutely identical with that of enteric fever at the present time. That is to say, that the bulk of fatal fevers in India is, and has been from the first, enteric in nature. The numbers of cases occurring, *and officially returned as enteric fever*, has gradually and progressively increased; and this is due to the gradual improvement in the differential diagnosis of fevers, rendered possible by thermometry and other aids of comparatively recent date, rather than to an actual increase in the number of cases occurring in India. Bryden, in his Reports in the A.M.D. blue-books up to 1876, stated that it was "a matter of popular observation, that no regiment or battery escapes enteric fever in its first year's service in India, whatever cantonment be selected." Of seventy-three regiments and batteries which arrived in India between 1871 and 1877, nine only remained free from the disease in the first year after landing. He compares the disease's prevalence at various stations per 1000 of average strength: home, ·99 admitted, ·24 died; Bengal, 3·75 admitted, 1·53 died; Gibraltar, 4·04 admitted, ·89 died; and Malta, 4·72 admitted, 1·57 died.

Between 1870–77 the admission-rate fluctuated between 2·8 and 4·6; and the mortality per cent. of those attacked varied from 50·88 to from 39 to 49, or a mean of 43·73.

In 1878, 132 deaths from enteric fever were reported, *and* 90 *occurred in men under twenty-two months' service in India.* In 1879 and 1880, the admission-rates were respectively 8 and 7·9 per mille, and the death-rate of those attacked attained a mean of 44·72 per cent. During 1881, the admission-rate fell to 5·6, but there were 47·26 per cent. of deaths; and for 1882 and 1883, the admission-rates were 6·2 and 7·7 per mille, and the death-rates 2·55 and 2·4 per mille.

In 1885, the percentage of admissions for ague remains unchanged, but the increase from enteric fever is exactly compensated by a decrease in that of remittent and simple continued fevers.

In 1886, there occurs in the Report of the Surgeon-General with the Government of India a summary of the views of different medical officers serving in India as to the frequency of enteric fever there:—

(1.) Enteric fever is the principal fever of India, along with ague and simple continued fever, and some of the cases returned as simple continued fever are in reality mild cases of enteric fever.

(2.) There are separate diseases called enteric, remittent, simple continued fevers, and ague, the differential diagnoses of which are, or will be, possible through patience, perseverance, and increasing knowledge.

(3.) There are cases which can easily be recognised as ague, enteric, remittent, and simple continued fevers; but difficulty of diagnosis, when it exists, corresponds to the reality that these affections shade off into one another, being due to one "causa constans," influenced by different circumstances.

(4.) There is little or no European enteric fever, but a fever with catarrhal follicular inflammation and ulceration of the bowel is common,—*an* enteric fever, but not *the* enteric fever.

In 1889, the death-rate from enteric fever increased by 2 per 1000 over that of 1888, and the admissions by over 9 from 1888. Bengal comes highest, and Madras lowest, for both admission and death-rate,—Bareilly, in the North-West Provinces, being the cantonment giving the highest rates, and Fort-William in Lower Bengal the lowest, in the Bengal Presidency. Mhow, in the Bombay Presidency, came next to Bareilly in the height of its ratios.

I have dealt as shortly as possible on this point here, as I have elsewhere given full details and statistical tables illustrative of the prevalence of enteric and the malarial fevers of India.

Etiology.

At the outset, I must state that it is not my purpose to discuss here the current views of the etiology of typhoid fever *as seen in Europe*. It is only the application of our knowledge to the totally different conditions of Indian life that I propose to review and discuss at present. Under the head "Etiology" I propose to discuss:—1. The minor theories; 2. The climatic theory; and 3. The pythogenic and specific theories. We are here confronted with several minor theories, which call for very brief notice and criticism on my part, as they have already been thoroughly demolished by previous writers on the subject, notably by Surgeon-Major A. Duncan, M.D. Lond., F.R.C.S. Eng., of the Bengal Medical Service, in his work on *The Prevention of Disease in Tropical and Sub-Tropical Campaigns*, already referred to.

1. These *minor theories* are those with which the names of Stich, Sir W. Moore, Martin, Colin, and von Pettenkofer are associated.

Stich's theory asserts that the disease may arise by chemical and bacterial changes induced in the contents of the alimentary canal, "under the influence of disturbing causes." Laveran has demonstrated that "this theory has no precise facts to rest upon." But, though this may be true, yet the theory in part supports what we know about the transmutability of the bacterium coli commune, a point which will be discussed later in considering "the specific theory." W. E. Porter's theory, that the decomposition of the fæces in the intestinal canal of the patient produces the disease is so closely related to Stich's hypothesis that it may be mentioned here in conjunction with it.

Sir William Moore's theory asserts the non-specificity of enteric fever, and states it to be simply "a phase of fever;" and he goes on to state that all fevers belong to one genus, and to describe the eruption of enteric fever as "petechial," and also to assert its identity with those of typhus, purpura, and scurvy!

Duncan remarks *à propos* this theory and with reference to the last-mentioned statement, "It is surely enough, with regard to this theory, to point out that the last three eruptions do not disappear on pressure," and one cannot but agree with him that the other premises are equally fallacious.

Martin's "Vicarious Theory" ascribes enteric fever to the exhaustion consequent, in a longer or shorter time, on the prolonged stimulation of the hepatic function by tropical heat, by the excessive work thrown upon the liver by the lessened amount of oxygen in heated air, and the consequent respiratory modifications. He thinks that the "intestinal glands" assume a vicarious "and abnormal activity, supplemental to the hepatic insufficiency." This elementary function of the "intestinal glands" at length induces a "suppurative enteritis," he thinks, and the reason he ascribes for the assumption by the "intestinal glands" of this vicarious function is an "idiosyncrasal proneness of the glands to this abnormal function." This theory would, he thinks, explain the "occasional spontaneous origin (of enteric fever), and also its non-occurrence or rarity among the natives of tropical climates."

The premises here are incorrect, as Duncan has shown, for the solitary and Peyerian glands are alone affected, and are *absorptive* and not eliminative in function; and the gland lesions do not occur till the fever has been in progress for some days, whereas Martin would make the gland-process the starting point of the whole pathological condition. Moreover, Budd long since pointed out that the Peyerian lesions were in all probability merely the local expressions of a general state. "Again, the longer a man lives in India, the more deranged does his liver become, hence we would expect more 'vicarious action' of the 'intestinal glands,' and consequent enteric fever as age increased, but this is just what we do not find; nor is functional hepatic derangement a prodromal symptom of enteric fever." And, finally, natives suffer from enteric fever far more frequently than was formerly supposed; and it is quite incorrect to describe the enteric process as a "suppurative enteritis."

Colin's Transformation Theory.—Professor Leon Colin, of the Vâl de Grace, an eminent French army medical officer, holds that "paludal typhoid" exists, and is the result of the combined action of paludal and typhoidal elements,—well described by Surgeon-General Woodward's (of the U.S. army) term of "typho-malarial,"—and that many cases of malarial fever merge into typhoid fever. He also holds that "all acute febrile conditions, accompanied by a marked alteration in the secretions and by gastro-intestinal complications, may induce the spontaneous development of typhoid fever[1] (p. 276); and that, in such cases, it would be impossible during life to recognise the affection, for the two diseases have

[1] "De la Fièvre Typhoïde Pallustre," *Arch. Gen.*, 1879, t. i.

ceased to be distinct,—the remittent being transformed into enteric fever." [1]

He thinks he has proved that the body can receive, at the same time, two miasmatic agents; and, in connexion with this statement, it may be mentioned that many observers suppose a malaria-stricken patient to be specially prone to an attack of enteric fever, —a point which I shall endeavour to controvert.

There exists much evidence of a positive nature against this theory. Although it is true that the malarial and enteric fever poisons are in no respect antagonistic one to the other, yet there is in reality no evidence to indicate any causal or essential connexion between them. Enteric fever is certainly not a form of malarial fever, for it occurs anywhere in the plains of India, even where ague is unknown; and when the two diseases co-exist, they do not prevail contemporaneously, nor are those stations which are notoriously malarious specially remarkable for the prevalence of enteric fever,—in fact, the reverse often appears to be the case.[2]

Again, ague attacks men of all ages, while enteric fever shows a preference for young subjects, nor do any number of attacks of ague (except as those imply length of residence in India) confer an immunity from enteric fever. Moreover, quinine cures ague, and does not affect the course of enteric fever at all. There is, indeed, no more ground for allowing that enteric fever can be roused into activity by two or more sets of poisons than there would be for asserting the same of any other of the exanthemata.

Pettenkofer's ground-water theory is too well known to need describing here. Though supported by many eminent epidemiologists, I consider that Parkes practically demolished the theory when he pointed out that the other conditions, stated by Pettenkofer to be necessary as present *in addition to the level of the ground-water* (viz., a soil impure from animal impregnation, a certain temperature, and the entrance of specific germs), are, in themselves, all-sufficient for the production of a typhoid epidemic, quite irrespective of any help from such an accidental condition as the level of the ground-water.

2. *The Climatic Theory.*—This is a very much favoured hypothesis by many military medical officers, and as such will deserve

[1] While admitting the etiological duality of *certain* epidemics of typho-malarial fever, "il établit qu'il ne faut pas chercher en dehors de l'organisme malade les conditions pathogéniques de l'ulcération des glandes de Peyer dans une fièvre paludéene d'origine. Les arguments invoqués sont la banalité des lésions de l'entérite folliculeuse, et l'identité des circonstances étiologiques au milieu desquelles apparaissent les fièvres graves avec lésion intestinale." (Kelsch et Kiener, *Maladies des Pays Chauds*, 1889).

[2] The explanation of the curious fact that an intensely malarious area when drained and inhabited has yielded enteric fever cases is simple enough. Before drainage, the locality was too unhealthy to be inhabited to any extent. After drainage, a community has sprung up there, and the inevitable communal disease—enteric fever—has been imported by some immigrant in his person, or by "fomites," and thus has been disseminated.

the closest attention. *Bryden* stated that enteric fever was engendered by the exposure of immature constitutions to the conditions of tropical life. *Sir Joseph Fayrer*, in his Croonian Lectures in 1882, put forward the hypothesis, that while true enteric fever undoubtedly does occur in India, there is a group of cases which are directly caused by the perversion of physiological functions consequent on the landing of the European in the tropics,—"Geographical position and climatic influences, heat, moisture, organic decomposition, miasmata, and a variety of aërial and telluric conditions which are more likely than a specific cause in India." He would distinguish[1] between the two forms of enteric fever as "specific" and "climatic," and says that the diagnosis between them is very difficult. I cannot do better than quote Surgeon-Major Duncan again,[2] whose remarks on this subject are so convincing as to require no additional criticism of Fayrer's theory from me. He says, "In the collection of opinions forming the basis of this theory, we find scarcely any definite post-mortem accounts . . . . . and there is an absence of proof that the cases brought forward to illustrate it were not due to a specific or pythogenic cause. From an examination of the cases, the argument reduced to logical expression is the following:—Cases of ulceration of the intestine are caused, in warm climates, by a variety of aërial and telluric conditions. But enteric fever is accompanied by ulceration of the intestine, therefore enteric fever is caused by a variety of aërial and telluric conditions. Finally, Sir J. Fayrer himself condemns his whole superstructure, for he proposes to call the fever 'endemic' as distinguished from the 'specific' enteric fever, but Murchison remarks that it is *the* endemic fever of England, as it is of France and America. It is a peculiarity of the disease that it is endemic everywhere. . . . . Two conditions are necessary to establish Fayrer's views—(1), that the fever in any given case is really enteric fever; and (2), that all specific or pythogenic causes are absolutely excluded: at present, neither of these conditions have been established." And I shall presently show that the conditions for the origin and development of enteric fever, on the specific or pythogenic theory alike, abound in India. The logical and philosophical rule, to have recourse to known laws where they seem capable of explaining phenomena, rather than to seek in some new and totally unknown direction, is *au fond* sound. We must first have those conditions of heat, moisture, and soil which are said to be capable of producing the disease defined, and demonstrated as existing before we can make any progress.

In connexion with the theory of Bryden, that the fever is due to the exposure of immature constitutions to the conditions of tropical life, let us briefly glance at—(i.) the seasonal prevalence; (ii.) and

[1] *Croonian Lectures*, 1882, p. 224. [2] *Op. cit.*, p. 198.

distribution of the disease; and (iii.) the physical factors which are said to predispose the young soldier in India to the disease.

(i.) *Seasonal Prevalence.*—Though this varies to some extent, we are safe in stating that the two maximum periods of prevalence in Bengal are—(α) from April to May, (β) and from August to October.[1] The second quarter of the year generally yields most cases, and next the third quarter, in the Bengal Presidency; but in Madras and Bombay the third quarter is the special season of prevalence. One may here pause to remark on the strangeness of the fact that enteric fever—a disease so closely related to local conditions—should have two annual periods of excessive prevalence in one and the same place at times when the local conditions differ so greatly as they do during the hot and rainy seasons. Malarial fevers have a distinct maximum in the rainy seasons, on the other hand, but this does not tend to strengthen the identity of these fevers with enteric fever, but rather to indicate "the outcome of imperfect discrimination of distinct forms, enteric fever finding the local conditions present in the hot weather, and malarial fevers those during the rainy season, specially favourable to its development. Heat and moisture are the main characteristics of tropical climates, and both occur everywhere *except in the hill stations;* yet we find enteric fever equally prevalent in hill and plain stations alike at the same season." Hence there can be no causal relation between the conditions of heat and moisture and the prevalence of the disease, or we would find a preference for the plains' stations. Another noteworthy point is that the disease is *present in every month of the year,* and its maximum intensity is found not to coincide with the heat maximum, but to be attained prior to its culmination: it is *receding* when the heat maximum has been attained.

(ii.) *Distribution of the Disease.*—No station is exempt from enteric fever, for it equally affects the cool hill climate; the warm, dry plains of Central India and the Punjab; and the warm, damp sea-coast and plains' stations of Lower Bengal and Assam. Surgeon-Colonel Welch, F.R.C.S. Eng., of the Army Medical Staff, has pointed out a most significant fact.[2] He says that if we believe the climatic theory, we must also believe that at some level,—as we enter a climate similar to that of the temperate, or even arctic, zones,—we should leave behind us climatic enteric fever. But this is just what we do *not* do, for it occurs equally at the highest and lowest altitudes.

(iii.) The main *physical factors* predisposing to the disease are (α) youth; (β) recent arrival in the country (which concurs with "want of acclimatization"); and (γ) alimentary conditions.

[1] The malarial fevers have their maximum prevalence in September and October.

[2] *Enteric Fever*, 1883. H. K. Lewis, London. This work is without doubt one of the ablest monographs extant on the subject.

(α) This predisposing cause has been so fully recognised that we will content ourselves merely with its assertion, founding our belief on the evidence and statistics of Murchison, Collie, Louis, and others.[1] This is well exemplified by a reference to the official returns of the military medical services in India. The deaths *per mille*, and liability ratios *per cent.* for 1889, for instance, were, for men under 25 years of age, 9·81 and 64·5 respectively; from 25–29, 3·25 and 21·37; from 30–34, 1·83 and 12·03.

(β) *Recent Arrival.*—Andral long ago showed how especially prone medical students, coming from the provinces to Paris to study, were to be attacked by enteric fever; and Budd[2] has shown the same thing with regard to peasants coming from the country to reside in a large city; and Murchison,[3] Liebermeister,[4] and Louis,[5] statistically proved the fact.

The chance an arriver in an endemic centre of the disease has to seizure by enteric fever is very considerable, and this fact, along with age-susceptibility, seems to me to be one of the most important determining conditions. It is to this rather than to altered environment, and the physiological changes thereby induced, that I look very largely for an explanation of the excessive predisposition of our "boy-army" to enteric fever, not only in India, but all over the globe; and I hope presently to show that nearly every Indian station must be considered to be an active endemic centre of the disease. Conversely, those inhabitants of a locality who have resided some time there enjoy a relative immunity from the disease.

An average *percentage* of liability in India would be, during the first two years of service there, 64·12; during the third and fourth years, 24·59; and from the seventh to the tenth years, 8·52. Hence the first two years are actually the most dangerous and fatal periods of the soldier's Indian service.

Fortunately, the young soldier is compensated for this excessive proneness to the disease by his lessened chance of acquiring those tropical diseases which are so fatal to older soldiers, for the average expectation of life diminishes much more rapidly in India than in Britain (Don).

These factors of age and recent arrival do not *per se* predispose to enteric fever in India more than at home. It is the arrival in stations which are active endemic centres of the disease—active from the more rapid decomposition and distribution of fæcal

[1] It is curious to find Manzini recording a case in which he found typical enteric fever lesions in a seven months' fœtus! A few cases have been recorded of death from this disease during the first few weeks of life, but these are very exceptional. Trousseau, on the other hand, recorded cases of undoubted typhoid lesions in a woman aged 64, Wilk in a woman aged 70, and other observers at 72, 86, and 90 (Murchison).

[2] *Lancet*, Oct. 29th, 1859, p. 432.

[3] *Continued Fevers of Great Britain*, 2nd edition.

[4] Ziemssen's *Cyclopædia of Medicine*, vol. i.

[5] *Recherches sur la fièvre typhoïde*, 1er. edit., t. ii., p. 375.

particles, with or without the specific germs—that brings about such an excessive prevalence of the disease in India, just as Louis, Chomel, and Andral showed was the case in Paris.[1] "Want of acclimatization" must be read, in this instance, as want of the immune condition conferred by residence in an endemic locality; in its ordinarily accepted sense it certainly does not confer immunity from the disease, as it would do if the cause was climatic. Moreover, rapid subjection to tropical heat does *not* augment the disease-ratio, and it seems immaterial whether a corps arrives at the beginning of the cold season or not, so far as the prevalence of enteric fever in it is concerned.

Thus, the only condition superficially comparable to acclimatization is the immunity obtained by long residence in an endemic area, but this is really quite irrespective of climate, as it holds equally true for temperate as for tropical climates.

(γ) *Alimentary conditions of the individual predisposing him to the disease's incidence.*—That meat-eating and spirit-drinking races are specially liable to enteric fever seems a well-recognised fact. The hill-tribes of India and the Gurkhas from Nepaul are great sufferers from the disease as compared with the vegetarian plainsmen. And the European soldier is well known to consume a great deal more meat—generally tough, because recently killed—and beer than is able to be borne by him in a tropical climate. Beer is a drink familiar to him at home, readily accessible in the canteen (where no spirits, however, are sold), and is, besides, a "long drink,"—a necessary qualification for favour in a "thirsty country" like India.

It is not too much to suppose that this has an important predisposing effect by producing intestinal catarrh from the irritation of the gut by imperfectly digested and fermenting food-products, and by choking the lymphatic system of the gut with these. But that such conditions can lead to the spontaneous development of the disease (except, perhaps, by conversion of type of the bacteria of the gut) I think is not proven.[2]

[1] I believe that this accounts for the idea, prevalent among old Anglo-Indians, that it is injudicious to come home frequently from India. This seems true enough, for such rapid changes of environment must throw a great strain on the accommodatory physiological functions, and break the immunity acquired by residence.

[2] In chronic lung disease—especially if a destructive process—the Peyerian glands atrophy, previously enlarging and caseating, and this is accompanied by catarrhal inflammation of the mucosa of the gut, hence giving rise to one of the forms of phthisical diarrhœa. In chronic renal and hepatic disease, in syphilitic or malarial cachexia, and even in simple starvation (as was shown in the Indian Famine of 1876–7), marked Peyerian atrophy has frequently been observed. This confirms the notion that invalids were immune from enteric fever to some extent. Liebermeister asserted that pregnant and puerperal women, and those nursing infants, enjoy a relative immunity, though Nathan Smith (*On Typhoid Fever*, Baltimore, 1832) states, on the contrary, that more women succumb to it because of its appearance during pregnancy or in the puerperium.

The holders of the climatic theory lay great stress on the *supposed* immunity of natives to enteric fever. This, however, is a mere question of facts, and the facts are decidedly against the climatists. I have before referred to Ewart's and De Renzy's evidence on this point, which is amply confirmed by Sir Guyer Hunter, M.D.,[1] of the Bombay Army, who states that, in his long and ample experience, he has observed that no race or sect is exempt from the disease, and that he has frequently treated cases of the disease in Hindus, Mohammedans, Parsees, etc. Dr Scriven[2] amply confirms this, and De Renzy gives perfect histories and pathological reports of cases occurring under his notice in the Rawal Pindi Jail in 1869.

Prof. Chevers in Bengal, Crombie in Burmah, O'Brien in Assam, and many observers in Madras, have reported cases of the disease among natives. Indeed, Hirsch shows that there is no racial exemption throughout the world from enteric fever. Surgeon-Major Ranking, of the Bengal Army, states that natives suffer far more than Europeans, who boil their drinking-water and avoid all sources of fæcal contamination, for the native community is notoriously careless about the source of their cooking and drinking-water, and so this is just what we would expect. Drs Cleghorn and Holmes, of the Bengal Army, state that the *apparent* rarity of the disease among natives is due to the fact that most of them pass through an attack in childhood, which passes unheeded by the parents as "feverish diarrhœa."

We know that, in Europe, enteric fever is a very frequent, though not at all a fatal, disease among children,[3] and so the same, *à priori*, might be expected to be the case in India.

A final and most conclusive proof of the total inadequacy of the climatic theory to account for the causation of the disease is the case of Fort Asigarh, in Central India.[4] The conditions there present are practically perfect preventatives of the prevalence of enteric fever. This fort is situated on an isolated rock, and there are no native villages anywhere near it, and it is far distant from any railway-station. The water is rain-water collected in well-constructed, carefully-guarded and inspected tanks. The conservancy arrangements consist of the dry earth system, the ordure being collected and lowered from the fort walls to the plains lying a considerable distance below, where the animals from the adjacent jungle are said soon to dispose of it entirely. The food and milk-supplies are closely supervised. The consequence was that a large European garrison, composed of young soldiers at the most susceptible ages, had *no* cases for many years of the disease,

[1] Late Professor of Medicine at the Grant Medical College, Bombay.

[2] *Ind. Annals Med. Science*, April 1857, p. 512.

[3] Eustace Smith, *On Disease in Children*, 3rd edit., 1886.

[4] Reported by an Army medical officer some years ago in the *Lancet*, though I unfortunately cannot give the reference.

although all the tropical climatic conditions were present, and malaria evidenced its presence by attacking the men in the form of ague. The specific cause was excluded, hence the disease was absent.

3. *The Pythogenic and Specific Causes.*—We have here the causes either of which seem best fitted to explain the etiology of the Indian disease, being most in accordance with observed facts, and with our most recent knowledge of the means of successfully preventing filth-diseases. The climatic theory has a fatalistic tendency since we cannot materially modify climatic conditions. The pythogenic and specific theories luckily have the same practical outcome,—the rendering innocuous of human dejecta, which are the principal media of the disease's spread.

The Pythogenic Theory of Murchison and Jenner asserts that the zymotic exhalations from normal human or even animals' stools produce, after reception in the human body, enteric fever,—the term "pythogenic" signifying "born of putrescence." Murchison considered the poison to be some decomposing animal product.[1]

The pythogenic theory practically asserts the *de novo* origin of a specific disease from either the genesis of a specific microphyte, or from the continued action of the products of fermentation of the food-stuffs ingested. The *de novo* theory I hold disproved by the arguments used in the demolition of Liebig's theory of "spontaneous generation:" *ex nihilo, nihil fit.*

Murchison's pythogenic theory has, however, received apparent confirmation from the observations of Gabriel Vallet,[2] just published, and to be presently discussed. We may allow that the fermentation of fæcal products derived from the external surroundings of the individual may be nocuous by the manufacture of new chemical compounds, or by providing suitable soil and nutriment for the development, within the intestine, of saprophytic or pathogenic organisms. Most animal alkaloids which are produced from albuminous decomposition belong to the group of leucomaines of the muscarine type, and tend to produce diarrhœa, and should these bodies predominate over those of the atropine group (which are frequently synthetically produced at the same time, and which physiologically counteract the muscarine group), diarrhœa would be produced, and the gut would be converted into a *locus resistentiæ minoris,* where the specific organism of enteric fever would find congenial surroundings for its growth and development. Bouchard has shown that the ptomaines normally present in urine are notably increased in enteric fever, and belonged to the muscarine group. It is not going too far to suppose that at least a portion of this increase came from the increased production of animal alkaloids in

[1] A useful aphorism might hence be coined,—"Malaria from decomposing vegetable, and typhoid from decomposing animal, matter."

[2] *Le Bacillus Coli Communis dans ses Rapports avec le Bacille d'Eberth et l'Etiologie de la Fièvre Typhoïde.* Paris, 1892.

the gut, as well as from another conceivable source—the tissues generally, to which the organisms had gained access by the blood-current.[1]

The specific theory has now been arrived at by a process of exclusion, but we would not deny the possibility of the truth of the pythogenic theory; for even in the case of the specific theory we have not reached "the end of the passage," as Koch's postulates have not been satisfied, in the opinion of a host of our most eminent bacteriologists, for it is yet held by a large majority that animals appear to be immune from human enteric fever.

By most bacteriologists the "bacillus typhosus" of Eberth (discovered by Eberth and Klebs independently of one another in 1880, and confirmed by Koch, Coats, and Gaffky) is accepted tentatively as the *causa constans* of the disease. One of the most recent utterances on the subject well describes the present position. Dr Burdon-Sanderson, in the *British Medical Journal* for November 1891, states that "the constancy of the observed relation between the occurrence and distribution of the bacillus (typhosus) seems to leave no doubt as to its etiological significance." This organism has been found "numberless times in both the stools and organs and tissues of typhoid patients."[2] Its property of exciting suppuration has been fully recognised. Rodet and Roux first cast doubts on the specificity of Eberth's bacillus in 1889, and state that the "bacillus coli communis"—which is constantly present in healthy stools—is the true specific agency in enteric fever. They found only it present in the pus of a localized peritonitis in an enteric fever case, and in another case found it in multiple hepatic abscesses which were present. They could not find Eberth's bacillus in the stools of patients during an enteric epidemic "on repeated examination," but found the bacillus coli instead, and almost in pure cultures.

[1] The supposed spontaneous origin of enteric fever in isolated spots has always been a strong argument of the pythogenists. But it seems to me that it is practically impossible to exclude all possible sources of the introduction of the specific poison. Dr Cayley has shown that typhoid stools retain their infective properties for at least nine months after having been buried, and probably longer. Mild, unsuspected, or ambulant cases may visit an isolated country house months before a case occurs there to direct special attention to the question, and it is well known that such cases are as competent to spread the disease in a most virulent form equally with dejecta from very severe or fatal cases. In cases occurring at such an isolated country-house, nothing is commoner than to hear "that all possible sources of infection have been excluded," and that the origin must hence be *de novo*. This arises from the inability to trace with the eye the continuity of the chain whose connecting links are known to be invisible. To conclude from this that no chain exists is ridiculous. "If the same evidence were true, it would prove the spontaneous generation of plants and animals" whose lineage it is often impossible to trace, but this impossibility is of no force whatever as an argument against their propagation by the law of continuous succession (Budd).

[2] *Brit. Med. Jour.*, editorial, June 11th, 1892, p. 1261.

The morphological differences between Eberth's bacillus and the bacillus coli communis are very slight, except that the latter grows more luxuriantly on culture media than the former. The most recent work on the subject has shown that Eberth's bacillus is not so commonly met with in typhoidal stools or infected water as had been supposed, and is, indeed, often absent from typhoidal stools altogether. Surgeon-Captain Bruce, M.S., of Netley, found that its appearance was coincident with the commencement of ulceration of Peyer's patches.

There is much probability that the bacillus coli communis acquires pathogenic properties under certain conditions in man. Tested on animals, both organisms have precisely the same effect; but the bacillus coli cultivated from water-closets is much more virulent than that isolated from healthy human intestines (*op. cit.*, p. 1262), and appears while passing through the human gut to undergo certain modifications approximating it in character to Eberth's bacillus. The question of the identity of the typhoid germ is at present unsettled, but we may proceed to apply the specific theory to the conditions of life in India, as being the theory for which most clinical and bacteriological evidence exists at present.

The *water-supply* in India is generally taken from wells sunk in the alluvial clay to a sandy water-bearing substratum, which everywhere underlies the surface soil at a depth of from 20–120 feet. The water is raised by leather buckets, and then emptied into a receptacle called a "mussuck" (made of a goatskin stitched together so as to form a pouch with a very narrow mouth, and which is carried by the water-carrier, or "bhisti," slung on his back). Neither the bucket nor the mussuck can be cleaned after use, and the mussuck is used always to convey water from *different* sources intended for different purposes,—the "clean" water for cooking and drinking, and the less clean for washing, watering horses, or for the garden, etc. Hence drinking and cooking water *must* suffer contamination from such receptacles, even supposing it to have been in the first instance undefiled, which unfortunately can rarely be counted upon. The task of cleaning out a well is a very difficult one, and one which is rarely properly carried out. The wells are often uncovered, and all sorts of filth may get in,—dead bodies of all sorts being frequently found. Again, on recovery from an illness the native indulges in a "bath of recovery"—a thanksgiving ceremony—and this is performed most conveniently and pleasantly by him at the well-mouth. Here he washes his body and clothes, part of the washings finding its way back into the well, and the rest polluting the soil round the well-mouth. The soil through which the water percolates to feed the springs and fill the wells is generally soaked with the excrement of ages, and we can easily imagine what havoc an ambulant or other case of enteric fever may occasion by defæcating in the neighbourhood of a well. The wells are examined *chemically*, but *not* bacteriologically, twice a year; and so the

delusion may be indulged in that wells are yielding pure water when the reverse would be shown by bacteriological examination.

Again, the water, if filtered, is more likely to come out worse than it went in, for the almost universal filter is one composed of three earthenware pots placed one over the other, only the middle one of which contains the filtering material,—a few lumps of wood charcoal and a quantity of sand (gathered generally from the nearest river bed—a favourite place for natives and cattle to defæcate—and placed in the filter without any previous washing). If water is boiled and *then* filtered, it is easily seen how misleading and futile the whole process is.

Milk may be contaminated by being watered with tainted water; by standing in a house whose atmosphere is contaminated by effluvium from a cess-pit, ordure-trench, or other place where enteric stools are present; or by the soiled garments of an enteric fever patient.[1]

Food may be similarly infected by being washed or cooked with tainted water, or by a foul atmosphere, or by flies (coming from ordure-trenches with the fine hairs of their legs soaked in loose enteric stools) crawling on the articles of food.

The *kitchens and cooking arrangements* are occasionally undoubted sources of food contamination. The water used in cooking may be tainted with fæcal products from a typhoid case; and the kitchens are the favourite *points d'appui* of the cook's friends, who come from the bazaar clad in rags or clothes which are veritable "fomites." The cooks and their mates generally cook with dirty hands, and while wearing filthy clothes. It is therefore not difficult to trace the source of much disease to the contamination of food lying cooked or uncooked in the kitchens under such circumstances. For the weekly nspection of regimental cook-houses—made at a known time by a medical officer—everything is "spick and span," only to be succeeded by a speedy relapse to the "old order of things," however.

The native does not recognise the possibility that water, drawn by him from the filthiest source, may have serious effects on the health of those he cooks for. Should any untoward result arise from the consumption of water containing cholera or fever-germs, he ascribes it to "kismăt" (fate), and as beyond human control!

The *air* respired, especially in dust-storms, must be frequently laden with particles of fæces, swept up from the surface of the ground, and containing in or on them the specific typhogenic germs. The air in the bazaars or native quarters is generally to be avoided, for these dwellings stand on soil soaked for ages past with the excrement—specific and non-specific—of the inhabitants; and the neighbourhood of the houses too often is cumbered with heaps of ordure and cess-pits. The effects of such soil on the

[1] That cows fed on garbage or contaminated grass can cause the disease has not been proved as yet.

water which percolates through it on its way to some source of drinking-water—well or spring—can readily be imagined.

The *ice and aërated waters*, so freely consumed in India, are great sources of infection, as they are too often made from water which is not even previously boiled before use, though its condition may have been still further vitiated by filtration, which has already been shown to be very harmful.[1] Food and sweatmeats bought in the native quarters are often contaminated either by their manufacture from contaminated articles or from subsequent contamination by the filth and the foul air of the native house.

Prostitutes (native) also spread a good deal of zymotic disease, as can readily be understood, and the drinks and food often consumed by the European soldier in their houses are frequent sources of the disease.

Even *swimming baths* have been accused of helping to spread the disease. These exist in most barracks, and are completely refilled twice a month, while one-seventh of their bulk is daily run off, and a corresponding amount of fresh water run in. If this is neglected, the water becomes foul, for two or three hundred men generally bathe there daily; and the danger produced by a mild or ambulatory case of enteric fever bathing—as has not unfrequently occurred—needs only to be indicated to be appreciated.

Clothes which have been washed (as they invariably are) by native washermen (dhobis) may become the means of spreading the disease, either by contamination from being washed in filthy river-side pools—the usual place used by the "dhobis"—or by being stored, previous to being returned to the owner, in native houses situated on, and surrounded by, ordure-saturated soil; or by being washed along with the clothes from a case of enteric fever in a native or European. The washermen are quite devoid of knowledge of the danger of these proceedings, as may be readily understood.

The *conservancy system* in vogue universally (except in Calcutta and Bombay, where the water carriage plan is carried out) is the

[1] There is an impression that total abstainers suffer more in India, and this has no doubt arisen from the fact that beer-drinkers consume beer made from pure water exported from Britain or got from the hill-breweries in India,—for, generally speaking, the hill-water is good. The abstainers, on the other hand, drink aërated waters manufactured on the spot with water too generally foul. At Kamptee, 27 men—*all abstainers*—in one regiment, and all from different barracks, were attacked with enteric fever. The latrines were in good order, and the drinking-water was from a carefully-guarded well, and was passed through a filtering tank, and afterwards refiltered in barracks. It was found that the water used in the "recreation-room" by the abstainers was taken from a different well, which was thought good enough for cooking-purposes, and which was found situated close to the site of a former latrine, and having a surface drain from a wash-house passing within four feet of it, from which there were signs of percolation into the well. The water yielded an excessive amount of organic matter on analysis. The well was closed and the fever ceased.

dry earth system. This is generally very well carried out in the case of regiments, the only flaws I have observed being,—(1), The leaving uncovered of the iron receptacles into which the ordure is collected from the privy-pans; (2), The occasional leakage of the carts employed to remove these receptacles to the trenches where the ordure is buried; (3), And the bringing back earth from the neighbourhood of the trenches (for use in the privy-pans) in these carts. In the latter case, the earth may be contaminated by loose fæces or by urine (impregnated, perhaps, with some specific poison) which has got spilt from the receptacles into the cart used to remove them. The trenching is generally extremely efficiently performed, and the area is inspected weekly by a medical officer or some member of the Cantonment committee, or other competent person.

The *arrangements in private houses* are not nearly so efficient. The chief points I have noticed are:—(1.) That no earth is used after the privy-pans have been used; (2.) The receptacle into which the privy-pans are emptied may be in a most insanitary condition—being left uncovered, or placed in an out-house close to the cook-house or dwelling, to save the "sweeper" (under whose charge the conservancy arrangements of the house are) the trouble of carrying the privy-pans a longer way to empty them; (3.) Urine may be poured out near the house to save the trouble of carrying it to the iron receptacle and there emptying it; (4.) The iron receptacle itself may never be cleaned, or kept coated with cold coal-tar; (5.) And it may not be regularly emptied every time the ordure-cart comes round to remove the night soil to the trenched area; (6.) Again, water instead of wood-ashes may be—and generally is—used to clean the privy-pans, and this operation is generally carried out immediately outside the bath-room door, and the washings sink into and pollute the surface soil; (7.) The conservancy arrangements of the native servants are rarely inquired into by householders, with the result that servants empty their bowels and bladders round about the cook-house or near the dwelling-house; (8.) When a trench is dug for their use it is generally neglected, and not filled in weekly and a new one dug, and hence it gets so foul that they go anywhere but to the trench to relieve the "calls of nature."

"Heat produces rapid putrefaction of most animal substances, and hence fæcal emanations quickly follow. Great heat also desiccates ordure, and causes its reduction to a fine powder, on which the wind can easily act and diffuse into the atmosphere." Dust storms are very frequent during the hot season; and, during one of these, one cannot fail to inhale a very large amount of dust *plus* fæcal particles, for the native defæcates everywhere about a station—a fact rendered too evident by the senses of sight and smell—and in the hot weather there is no rain to dissolve and carry this ordure below the surface, hence aërial contamination ensues.

Coincident with the great heat the water-supply lessens and becomes more concentrated, hence a much larger dose of enteric poison relatively is imbibed in the water drunk from a well contaminated by enteric dejecta. Again, more water is drunk in the hot weather because of the great heat which prevails; and the heat produces laziness on the part of the water-carrier, who tends to go to the nearest water-supply, irrespective of its quality, instead of going further, perhaps, for a better source. The European, in the hot weather, is also much more inclined to carelessness and indifference as to the source of his water-supply.

Later, when the rainfall occurs, moisture is added to heat in assisting putrefaction of surface impurities, and in assisting germs to develop which till then were perhaps latent. The rainfall also mechanically conveys the germs, etc., into the water-storage places, and brings it by this medium into contact with its favourite nidus —the human Peyerian and solitary glands.

The disease is often contracted on railway journeys, in rest-camps, and on the line of march, by the men's incaution in drinking native-manufactured aërated waters (made from foul water too often), and by drinking from roadside springs or wells, as well as by consuming contaminated milk, butter, ice, and various articles of food.

In conclusion, one may add that it would indeed be remarkable if India alone suffered from a form of enteric fever etiologically distinct from that prevailing throughout the world. The experience elsewhere of the close connexion of enteric fever with local filth causes has been so constant, and the sequence of events so unmistakable, as to make it impossible for us to disregard such experience, and adopt a climatic cause. The close connexion of the native and his surroundings with the European population shows that the asserted means of transfer are the most probable source of the disease in India. We grant this freely in the case of all the other exanthems: why exclude enteric fever?

When a filth history seems absent, we may feel certain that the cause has not been traced, and this is not to be wondered at, considering—(1.) The wide area over which the men contract the disease; (2.) The many ways of communication and the practical impossibility of tracing any one after a period of two or three weeks has elapsed, during which the disease has been incubating in the patient. As Corfield has said, "It would, indeed, be very wonderful if we could trace every case of enteric fever to a previous case." The men suffer most from the disease; and officers and their wives, and soldiers' wives and children, much less. The reason is, that the men spend a great deal more time wandering about the native bazaars, and are often more careless as to their diet and as to what they drink. Thus, an epidemic in the 33rd regiment at Kamptee was confined to the single men *alone*. Here the disease occurred precisely in the class indicated who subject

themselves to the danger of contagion outside barracks, for generally "Indian cantonments are oases of cleanliness situated in deserts of filth."

Prophylaxis.

The first point to consider is *the effectual disinfection of the dejecta of enteric fever cases*, which are the medium by which the disease is spread mainly. The ideal plan would be destruction by heat, and most conveniently by an open fire. A series of specially constructed mill-board trays with a glazed interior, 9 ins. × 9 ins., and having a rim 3 ins. high, which should contain a convenient amount of sawdust or other readily inflammable absorbent, would seem best suited for the purpose. The motion, after being passed into such a receptacle, would then be placed, *mouth downwards*, on a *clear-burning* fire, so as to minimize the risk of sublimation of infective particles (which might possibly fall on, and infect, articles of food, etc.), before they had been rendered sterile by the heat. Such receptacles might be very cheaply manufactured, if the demand for them was created. They would be light, easily slipped under a fever patient, like a bed-pan, and would do away with all the risks attendant on the system at present in vogue, as well as saving the nurse much trouble. Failing this, of course, the present system may be used of passing the stools into a bed-pan, containing two or three teacupfuls of disinfectant solution,—preferably a coloured and acidulated solution of perchloride of mercury,—prepared according to Parson's formula: Hydrarg. perchlor., ℥ss.; acid hydrochlor., ℥j.; aniline blue, grs. v.; water, 3 gallons.[1] The motion should then be thoroughly mixed with the solution by stirring with a chip of wood (to be immediately afterwards burnt) so as to insure the contact of the disinfectant with all portions of the dejecta. The bed-pan and contents should then be allowed to stand, covered, for at least an hour, to allow the disinfectant to act well (for Wynter-Blyth has experimentally shown that the degree of disinfection is in direct ratio to the period of subjection of the infective material to the disinfectant's action). The stools should then be buried in a separate trench, and not consigned to the trenches used for burying healthy human ordure.

The usual fever-room precautions must be rigidly carried out, such as immersion in pails of corrosive sublimate or bleaching-powder solutions, and subsequent boiling, of all sick-room linen, etc.; and for aërial disinfection of the room I would recommend numerous small wooden boxes with perforated lids, containing "metallic iodine," or a solution of carbolic acid, one part, and ether, two parts, may be placed in saucers about the room, or even M'Dougall's powder might be used for the purpose.

No mention has been made of the measures to be adopted where

[1] If corrosive sublimate is not available, a 5 per cent. carbolic lotion may be used. The sulphates of copper, iron, or zinc; aluminium or zinc chlorides, or lead nitrate, have all been found unreliable.

(as in Calcutta and Bombay) water-carriage of sewage obtains, for the same precautions should be carried out as rigidly there as where only the dry earth system is in use; and the burning of dejecta, where practicable, should preferably be carried out; or failing that, the dejecta should be subjected to the measures detailed before, previous to consigning them to the sewers.

All drinking-water used in the house where the case has occurred should be boiled, cooled, and reboiled (in imitation of Tyndall and Koch's "discontinuous sterilization" process), and then placed in receptacles well washed out previously with boiled water, and kept scrupulously clean and efficiently covered.

The nurse should wear light-coloured washing dresses, which should be treated like the sick-room linen; should not mix with the healthy people in the house; and for her own protection, be most careful—(1), not to eat or drink anything which has stood in the sick-room, but to take her meals in a separate room with previously washed hands and face; (2), when washing her hands, after attending to the patient, never to splash the water up on her face, but to wash her face in a *fresh* basinful of water; (3), and never to "flick" up the bed-clothes or patient's night-dress (previous to examination of the bases of the lung or abdomen by the physician, or when being "attended to"), for thus infective particles which have adhered to the linen may be detached and inhaled either by the medical attendant or by herself; and, finally, (4), she should never sleep in the sick-room. After the termination of the illness, the room should be limewashed and disinfected carefully; and it will be best in India to burn all mattresses where no means exist for subjecting them to steam disinfection; while all bedsteads should be most carefully washed over with strong disinfectant solutions if made of iron, but if wooden, they should be unhesitatingly burnt. A large waterproof sheet under the patient is a most useful adjunct during the treatment of an enteric case. In the case of the patient's death, the coffin should be thickly dredged with bleaching powder.

Coming now to the *general prophylaxis*, we shall find it best to consider it in the following way:—

Europeans on first landing in India should be shielded from the great danger of contracting the disease on their railway journey "up country," by eating contaminated food, fruit, etc., and drinking infected water, aërated or otherwise. I have frequently observed European soldiers drinking from the native "mussuck" at railway stations in India. A supply of water from a properly-selected source, and which has been previously boiled, should be carried in troop trains, in iced receptacles; and supplies of ice and aërated waters also should be carried, which have been made under close European supervision (as at present done at many messes in India), and from good water previously boiled. Food and fruit, purveyed by respectable dealers and of good quality, and guarded in every

way from the usual modes of contamination, could be with ease obtained; for there are numerous purveyors who would, for the sake of securing such a contract, submit their supplies to close official scrutiny by the medical and commissariat departments. The distribution of the food could easily be carried out under regimental arrangements in the troop trains; and under a modified system in ordinary passenger trains.

The advantages of locating regiments and drafts on first arrival in India in hill-stations for the first two or three years have long been urged on the Indian Government, and have to some extent been recognised by it. The barrack-sites in the hills are, as a rule, singularly free from fæcal and other organic impurities; and as a less cubic space suffices there than in the plains, the barracks can be of smaller size, and hence less costly. The water-supply in the hill-stations is generally good, and can be more carefully guarded against contamination than in the plains; for there is a relatively sparse native population, for the plainsman does not thrive in the hill climate. A very high standard of health might be maintained among the men, for much more exercise could be taken in a fresh, cool atmosphere, and at the same time more military exercises and training could be carried out than is at all possible in the plains' stations during the hot and rainy seasons. Troops proceeding on active service from hill-stations have always maintained their health and vigour on campaigns better, and have been able to do far more and better work than their malaria-stricken and heat-debilitated comrades from plains' stations. Lastly, the men would learn to take better care of themselves, and to accommodate their systems to the new surroundings, under conditions which would be undoubtedly the most favourable. Many of the existing hill-stations are by no means in the best sanitary condition, for in pre-sanitary times it was thought enough to send men to the hills, even though the most obvious sanitary precautions there were overlooked. These defects have been, and are being steadily remedied, however.

But if a regiment cannot be sent to a hill-station, how must we guard the soldier best from enteric fever in a plains' station? Jenner, Simon, Budd, and Hirsch consider that few points in the etiology of typhoid fever are so certainly proved as that it is most generally conveyed by drinking-water. Again, impure water, although it may not contain the bacillus typhosus, predisposes, in some measure, to the disease by irritating the gut. The cleaning out of wells should be closely supervised, for it is usually imperfectly carried out. The brickwork, faced with cement, forming the sides and parapet-wall of the well, should be periodically examined for holes in it, made by storm-water or otherwise, and through which surface-impurities or vermin may find their way into the well-water. All wells from which cooking or drinking water is taken should be securely covered, and orders given the water-carriers to immediately recover them after drawing water. All washing in the vicinity of

wells should be forbidden. We must try to educate the troops and people generally in India to go, or to send, for their drinking and cooking supplies to a source proved by careful and skilled chemico-bacteriological examination to be absolutely pure, and should enforce the necessity of twice boiling all water taken from any other source. The difficulty is to obtain a pure source, unless it is led from long distances into the cities; and though this would entail an enormous expenditure, it would repay Government in the longrun.[1]

The *drawing of water* from the wells should invariably be by means of zinc or cast-iron pails, which can be easily cleansed; and the leather buckets, at present so widely employed, should be only used for drawing water from wells set apart for the supply of the garden and stables; though, of course, the best available water should always have the preference for these purposes if sufficiently plentiful. All cooking and drinking-water should be drawn from the wells in metal buckets, in which it should be carried to the kitchen or house.[2] The mussuck should be entirely abandoned for carrying water for any purpose, as it cannot be cleaned.

Ice and aërated waters should alone be manufactured from boiled water, and from as pure a source as possible.

The *milk-supply* should receive constant attention. The cows should belong to some responsible body, such as the mess-committee or the "coffee-shop committee" of a regiment; or the civil surgeon (an officer invariably of the Indian Medical Service) should agree to supervise some system for the supply of good milk to the station.[3] The cows should be well selected, carefully fed and housed, and milked under the supervision of orderlies selected for intelligence and experience of the devices by which the native milkman defrauds the buyer.[4] The milk-cans should be carefully rinsed with boiling water previous to use; the cow's udder and the milkman's

[1] After boiling water intended for domestic consumption, it should be placed in a perfectly clean receptacle and covered, the impurities being allowed to separate by subsidence, rather than suffer risk of contamination by filtration by the *usual* Indian filter previously described. Of course, where a really good filter (capable of having its charcoal easily removed and purified by heating to a white heat from time to time) exists it may be advantageously used, but always preferably prior to boiling the water. Another important detail is the use of *drinking-water* when brushing the teeth, as the toilet-bottle is generally filled at the same time, and from the same source, as the ewers and baths.

[2] In India, the kitchen is almost invariably separate and at some distance from the house.

[3] This, I know, has been most successfully undertaken at Bareilly by my friend, Surgeon-Major J. Anderson, Bengal Medical Service, at present civil surgeon of Bareilly.

[4] These are chiefly three: (1.) Starting milking into cans containing some water already; (2) or by adding water later after the milking is completed; or (3) by running it in as fast as the milk by means of a small mussuck under the milker's clothes, from which a tube descends along the palmar aspect of the arm and hand and thus into the pail.

hands well washed; and it should be seen that, if the cow has a calf, there is nothing of the nature of a specific enteritis affecting the calf, for it is yet uncertain whether this disease is not closely related to human typhoid fever, as Dr Jas. Allan has declared it to be in his work entitled, *Is Enteric Fever a Cattle-Disease?* After the cans are filled with milk, they should be at once securely padlocked and removed to the ice-house (where such exists in a regiment), and subsequently distributed under close European supervision.

The ration-meat generally supplied by the commissariat is of comparatively good quality. *Vegetables*—such as tomatoes, lettuce, and others which are eaten uncooked—should be thoroughly washed *in drinking-water;* and all *fruit* used should be supervised, and all that is bruised discarded. All articles of food when standing on the table should be carefully covered to prevent access of flies and other insects, for the former especially revel in ordure heaps, from which they may come straight to, and land on, some article of food.

It has often been asked, "How are we to disinfect *butter and cream?*" When manufactured, these articles are not amenable to any sterilization process, but we can take care that their common source—milk—is sterilized by boiling before the butter or cream is made from it. This can easily be carried out in India in the case of private families, for the cows or goats supplying the milk are generally the property of the consumer. What is possible for the individual family is equally possible for officers', sergeants', and men's messes, where the details could be easily carried out.

The kitchen in India has generally an earthen floor, and the certainty with which soil contamination occurs (with consequent deleterious exhalations) should teach us to aim at providing each kitchen with a concrete floor. This is a cheap and very necessary reform.

The cooking operations should be supervised by means of regular and "surprise" visits to the kitchen by someone sufficiently competent and interested in the regulation of the irregularities of the Indian cook. Clean clothes should be compulsory for the cook, and should he prefer (as is very often the case) to cook in a semi-clothed condition, a preliminary good ablution of his hands and person should be insisted on.

More attention is required to the dietetic requirements of the soldier in India, and more particularly in the direction of diffusing a few simple general rules for his guidance. The adaptation of alimentary principles to the physical requirements—a lessened heat-production and energy out-put—is of prime importance where diseases of the abdominal viscera bulk so prominently as they do in the returns of our army in India. Many Europeans suffer from the delusion that large meat meals are necessary to "support their strength;" and that highly-spiced food, chutneys, curries, etc., are useful articles of diet. But the latter articles only tend to increase the

hepatic turgescence due to the joint action of tropical heat, lessened respiratory excretion, malarial poisoning (which increases hepatic hæmolysis enormously as well as congesting the liver in common with the other abdominal viscera), and diminished physical exercise.

The *aërial propagation* should be prevented as far as possible by enforcing strict cleanliness, in the neighbourhood of, as well as in, cantonments, of the surface-soil.

The *conservancy arrangements* for troops, as already provided for by the regulations, are excellent; and the rules issued for the efficient working of the dry-earth system are, if carefully carried out, quite sufficient. I have already directed attention to a few points in which danger may readily occur; the remedies here are sufficiently obvious. Another detail in the working of the system is, that iron scoops (used for throwing earth on the dejecta) should always be used in place of wooden ones, which are liable to absorb deleterious matters, and are less easily cleansed.

The *arrangements in private houses* are much more defective (*v.* page 289). White, glazed, earthenware privy-pans are in general use, which, after being used, are carried away and emptied. The pan is then washed with water. Now, this is clearly not the dry-earth system (which, however, it is supposed to be), for no earth is used here at all. The remedies are, obviously, to use sufficient dry earth to cover the motion, and to take care that woodashes, and not water, are used to clean the pans after use. The iron receptacle should be well coated with cold coal-tar weekly.

Finally, the *servants' conservancy arrangements* require much more attention. The trench set apart for their use should have plenty of dry, loose earth near it to cover the dejecta with; and a new trench should be dug every week at least, and the old one carefully filled in and left undisturbed for several months. The native invariably uses water for cleaning his person after defecating, and it is important that this operation be carried out so that the washings fall into the trench.

Surface and subsoil drainage should be thoroughly efficient, so as to prevent the stagnation and decomposition of animal and vegetable matter, thus lessening the number of "forcing beds" for the disease.

The washing of clothes should be supervised. For large bodies of people this can be done under Government arrangements, which should insure the purity of the water used for washing, and also of the tanks in which the clothes are washed; that the washermen's persons and clothes are reasonably clean when engaged in their work; and that the clothes, after washing, are stored in a place where they cannot be contaminated by soil-emanations, or by infected linen or persons. In private houses this can be still more easily done by insisting on the washerman washing the clothes on the premises, and in tubs provided by the owner, who would also see that clean water was used, and that the clothes were dried and stored in a proper place.

A high standard of *personal cleanliness* should be encouraged in the men; and morning and evening—at the latter time specially—outdoor sports and exercises should be "got up" with the view of keeping them away from the native villages and bazaars. The subject of the sanitation of the native quarters has been recently brought up by Miss Florence Nightingale;[1] the crusade against their terribly insanitary condition should be pushed to the utmost, for this is *fons et origo mali*. Unless consummate tact is employed, this is a matter which the vast majority of the native community may be expected to resent, as they are suspicious, ignorant, fatalistic, and utterly indifferent on all such questions, and thus the way must be felt with the utmost circumspection. We must begin by explanation of the necessity for, and advantage of, sanitary reform to educated and influential natives, who may be induced to use their influence with their co-religionists to further reforms in this respect. Native doctors, hospital assistants, etc., may do much good by helping to educate public opinion in this direction, and in preparing the way for such obvious sanitary reforms in native communities, as the introduction of good conservancy arrangements, and of a pure and sufficient water-supply (preferably led in from a distance), and the lessening of overcrowding and other insanitary conditions. Such simple changes as these will take long to effect in conservative, apathetic India; and it must be many years before even an approximately sanitary condition can be attained in native communities.

The desiderata which should constantly be kept before the notice of the medical officer in India are: the extirpation of filth as the fostering bed of the virus; the severance, so far as is possible, of the links of transference between the source of production and the young, susceptible soldier; the increasing of the age of soldiers serving in India, so as to bring them to that country when the age-susceptibility shall have materially lessened; and to reduce the frequency of change between India and England, by lengthening the service-period for India by offering greater inducements for time-expired men to re-engage for a further period of service in that country; to locate every European it is possible to in hill-stations; and to attack vigorously the insanitary conditions of those hot-beds of the disease,—the native quarters with their terribly insanitary surroundings.

[1] The condition of the native quarters must be seen to be realized. The houses stand on soil saturated for ages past with percolated filth; the conservancy arrangements are of the worst description, or conspicuous by their absence; overcrowding and want of ventilation are features almost universally present; and personal and domestic cleanliness is neglected; and epidemic and contagious diseases may exist within the native's house unsuspected and uncontrolled, for the zenana is a safe sanctuary for disease where the sanitarian cannot intrude. Within cantonment limits, however, supervision is possible by warning the householder that his house-yard or elsewhere will be inspected at a certain hour, and to remove his family for the time from the place to be inspected.

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